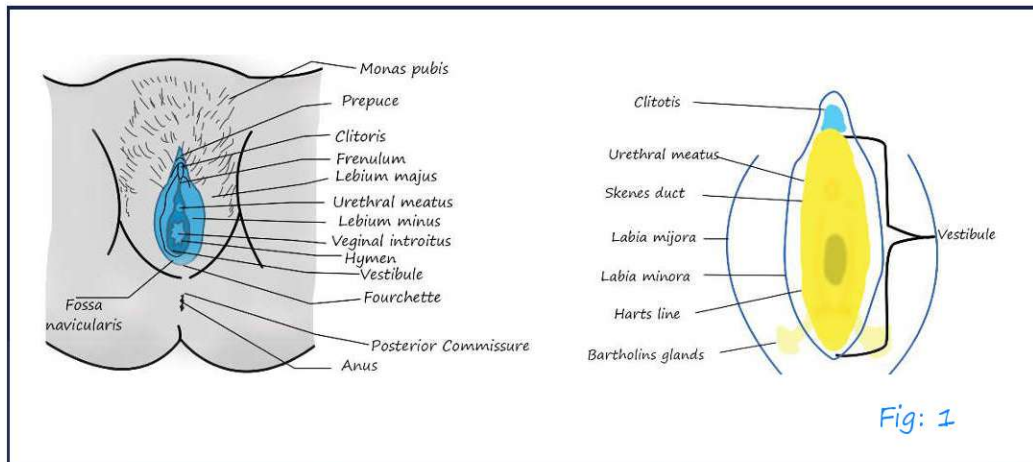


ANATOMY OF FEMALE GENITAL TRACT- I



Structures Include

- Mons Pubis
- Labia Majora
- Labia Minora
- Clitoris
- Hymen
- Vestibule
- Urethra opening
- Greater vestibular / Bartholin glands
- Minor Vestibular glands
- Paraurethral / Skene gland
- Mons pubis is thick pad of fat covered with hair (Sexual Hair)
- Hormone testosterone is needed for development of pubic / sexual Hair

Labia Minora

Homology of sexual organs

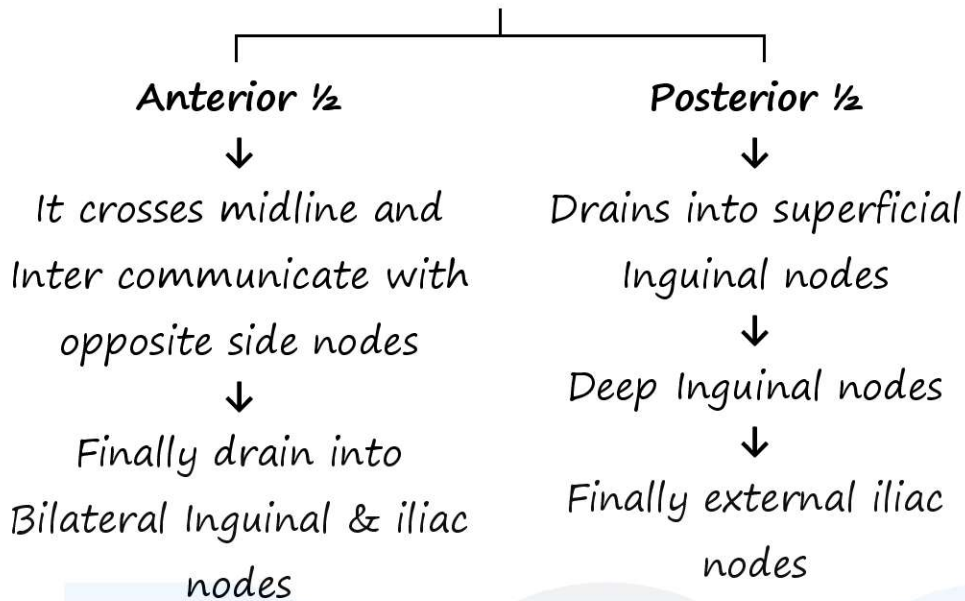
<i>Female</i>	<i>Male</i>
i. Ovary	Testis
ii. Para oophoron	Ductus deferens
iii. Paraurethral / Skene gland	Prostate
iv. Vagina (lower end)	Prostatic utricle
v. Clitoris	Penis
vi. Labia Majora	Scrotum
vii. Labia Minora	Urethral surface of penis
viii. Vestibular bulb	Corpus Spongiosum

Labia Majora

00:08:36

- It has thick pad of fat without muscle fiber. It is covered with skin having sexual hair
 - i. Homologous to scrotum in males
 - ii. Covered with sexual hair
 - iii. Skin: - Stratified squamous keratinized epithelium
 - iv. It has no muscle fibers
 - It has only fat and elastic fibers
 - v. Both sebaceous glands and sweat glands present
 - vi. Rich in lymphatic and venous plexus
 - vii. Lymphatics of labia majora

Lymphatics of labia majora



viii. Round Ligament terminates into labia majora at its upper border

Labia Minora

00:10:39

- This labia Minora in its upper portion divides into two, which enclose the glands including tip of clitoris, thin tissue folds medial to labia majora
 - i. Superiorly divides into 2

Upper – Prepuce
Lower – Frenulum
 - ii. Inferiorly joins to form FOUCHETTE
 - iii. Two different types of epithelium cover labia minora separated by **line of HART**
 - Medial to HART line – non-Keratinized squamous epithelium
 - Lateral to HART line – Keratinized squamous epithelium

iv. It is devoid of sebaceous & sweat glands

Clitoris

00:12:55

- i. Organ of orgasm & homologous to penis in male
- ii. Located between prepuce and frenulum,
- iii. Tip is called Glans
- iv. Normal length – should not exceed 1 cm, if $> 1\text{cm}$, it is called **clitoromegaly**, which is a sign of **Hyperandrogenism**
- v. Length of glans – 0.5 cm or less
- vi. Arterial supply – deep and dorsal artery of clitoris
- vii. Lymphatics – directly drain into Deep Inguinal lymph nodes
- viii. Venous – Rich in plexus, drain into dorsal vein of clitoris

Vestibule-

- Area enclosing all the openings in vulva

Boundaries

- Laterally – Hart line
- Medial – Hymen
- Anterior – Frenulum
- Posterior – Fouchette

Six openings

- Urethra
- Vagina
- 2 Bartholin glands
- 2 Skene / Paraurethral gland
 - Fossa Navicularis – Tissue between Hymen and Fouchette

Bartholin Glands

- i. Also called – Greater Vestibular gland
- ii. Gland size – 0.5 to 1 cm
- iii. Size of duct – 1.5 to 2 cm
- iv. Opening of Duct – 5'o and 7'o clock position

Para urethral Glands

00:18:27

- Also known as **Skene Glands**
- Paired glands – one pair on right, one pair on left along the inferior aspect of urethra
- Inflammation of glands –obstruction of ducts-
Urethral Diverticulum

Internal genetaila

00:19:08

Vagina

- i. Tubular structure extending from cervix to Hymenal opening.

- ii. It has anterior wall, long posterior walls and 2 lateral walls.
- iii. With Ectocervix, it forms fornices – anterior, posterior and 2 lateral fornices.
 - Posterior fornix is deeper due to long posterior vaginal wall.
- iv. Epithelium: Non-Keratinized stratified squamous epithelium.
- v. Externally vagina opens at Hymen, which is membranous structure with small opening to allow drainage of menstrual blood & fluid
- vi. Due to repeated intercourse or during process of childbirth, Hymen ruptures and it is replaced by Rudimentary structure – **Myrtiform Caruncles**.
- vii. Boundaries:
 - Anterior: Separated from bladder by uterovesical fold (uv fold) of peritoneum.
 - Posterior: Separated from rectum by Rectovaginal fold of Peritoneum.
 - Lateral wall: Up 1/8 – levator ani muscle

Length

- Anterior wall – 6 to 8 cm
- Posterior wall – 7 to 10 cm
 - It has mucosal folds called Rugae or Vaginal Rugosities
 - It has no glands

- It has smooth muscle, collagen and elastin

Blood Supply

- Upper vagina – Descending cervical / vaginal branch of uterine artery
- Posterior vagina – Middle rectal artery
- Distal part of Vagina – Internal pudendal artery

Lymphatic Drainage

- Upper 1/3 – Internal iliac, external iliac and common iliac nodes
- Middle 1/3 – Internal iliac Nodes
- Lower 1/3 – Inguinal lymph nodes

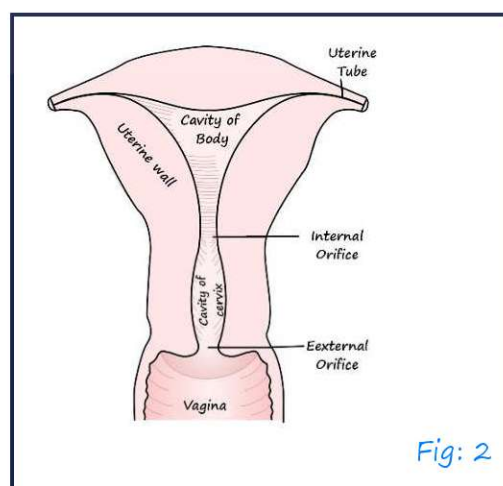
Uterus

- i. Main female reproductive organ presents between Bladder anteriorly and rectum posteriorly
- ii. Pear shaped organ.
- iii. Uterus – Body or corpus and Cervix
- iv. Length – Nullipara – 6 to 8 cm
 - Multipara – 9 to 10 cm
 - Weight of uterus – 60gms (non-pregnant stage)

Cervix

- i. Lower part of uterus
- ii. Lower most part of cervix protruding into vagina – Ectocervix, covered by external OS

- iii. Cervix - Supra vaginal / Endocervix with internal OS
 - Infra vaginal / Ectocervix with external OS
- iv. External OS corresponds to Ischial spines
- v. Internal OS corresponds to UV fold of peritoneum
- vi. Shape of Cervix - Before delivery → Oval / Round
After delivery → Transverse slit
- vii. Epithelium - Ectocervix - Stratified Squamous
 - Endocervix - Columnar epithelium
- viii. Junction of both Ectocervix and Endocervix corresponds to **Transformation Zone, which is more prone to ca of cervix**
- ix. Eversion of Cervix - when columnar epithelium of Endocervix exposes to vaginal PH. It is eversion of Cervix
- x. It has little smooth muscle, mainly consist of collagen and Elastic Fibrils, which helps to stretch cervix during vaginal birth



Ligaments of Uterus

- i. Uterosacral ligaments
 - ii. Mackenrodts / Cardinal / Transverse cervical ligament – **Main support of Uterus**
 - iii. Round ligament
 - iv. Broad ligament
- } **No role in support of Uterus**
- v. Ovarian ligament
- Blood supply to round ligament → SAMPSON'S artery^Q**

Blood Supply of Uterus

- i. Uterine Artery (Major part)
Ovarian Artery (Minor Part)
- ii. Ovarian Artery → Branch of internal illiac artery
→ Supplies mainly fundus of uterus and part of fallopian tubes.

Uterine Artery

- Branches into segmental / Arcuate artery



Radial Artery



Straight artery / Basal Artery → Supplies Basalis layer



Spiral Artery → Supply functional layer of uterus

- Functional layer of uterus – Superficial compact layer
- Intermediate spongy layer

Lymphatics of Uterus and Cervix

00:34:08

Uterus

- Internal iliac nodes – **Mainly**
- Para aortic nodes

Cervix

- Internal iliac Nodes

Nerve Supply

- Sympathetic – T₁₀ to L₂
 - Parasympathetic – S₂, S₃, S₄
- } Forms inferior hypogastric Plexus

Inferior hypogastric plexus divides into

- Vesical Plexus
 - Middle Rectal plexus
 - Utero Vaginal plexus
- Pain from uterus – T₁₀ to L₂
 - Pain from Cervix, upper vaginal – S₂, S₃, S₄ (Pelvic splanchnic nerves)
 - Pain from lower vagina – Pudendal Nerve (S₂, S₃, S₄)
 - Pain fibres during uterine contractions carried by sympathetic T₁₀ to L₁

Ovaries

- i. Paired, pearly white almond shaped structures
- ii. Dimensions – 3 x 2 x 1 cm
- iii. Ovary – Outer cortex – with follicles
Inner medulla – Blood vessels
- iv. Epithelium – Single layer of cuboidal epithelium
- v. Tunica Albuginea → Connective tissue surrounding the ovarian epithelium
- vi. Blood Supply – Mainly ovarian artery (90%) which is anterior branch of internal iliac artery
- vii. Uterine artery (10%) (Ovarian ligament)
- viii. Nerve Supply – Sympathetic – T₁₀
- ix. Parasympathetic – Vagus nerve
- x. Ovaries are homologous to testis in males developed from coelomic epithelium embryologically.

TUTORIALS

LOGIN FOR SUCCESS

FUNDAMENTALS OF REPRODUCTION

- Gametogenesis
- Semen capacitation
- Acrosome reaction
- Fertilization
- Implantation.

Gametogenesis

- Definition: The process of formation or maturation of highly specialized cells, spermatozoon in male or ovum in female before they unite to form a zygote.

Oogenesis

AfraTafreeh.com

00:03:03

- The process involved in the development of mature ovum.
 - i. Primitive germ cells take origin from yolk sac → begins at end of third week. ^Q
 - ii. Migration of primitive germ cells to the developing gonadal ridge is completed by end of 4th week ^Q
 - iii. Primitive germ cells are called oogonia. ^Q

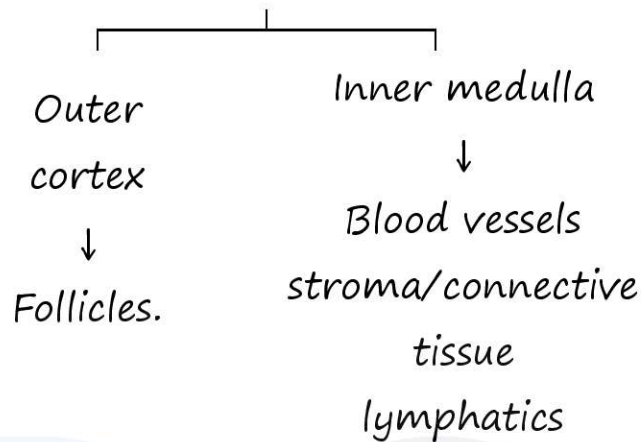
Oogonia

↓

Mitotic division.

Karyotype of oogonia – 46xx ^Q

iv. Majority of oogonia divide by mitosis, but few oogonia enter meiosis and are known as **primary oocytes** ^Q



- Meiosis begins between 12-20 weeks of intrauterine life
- v. All oogonia are replaced by primary oocytes at birth.
And no more mitosis takes place.

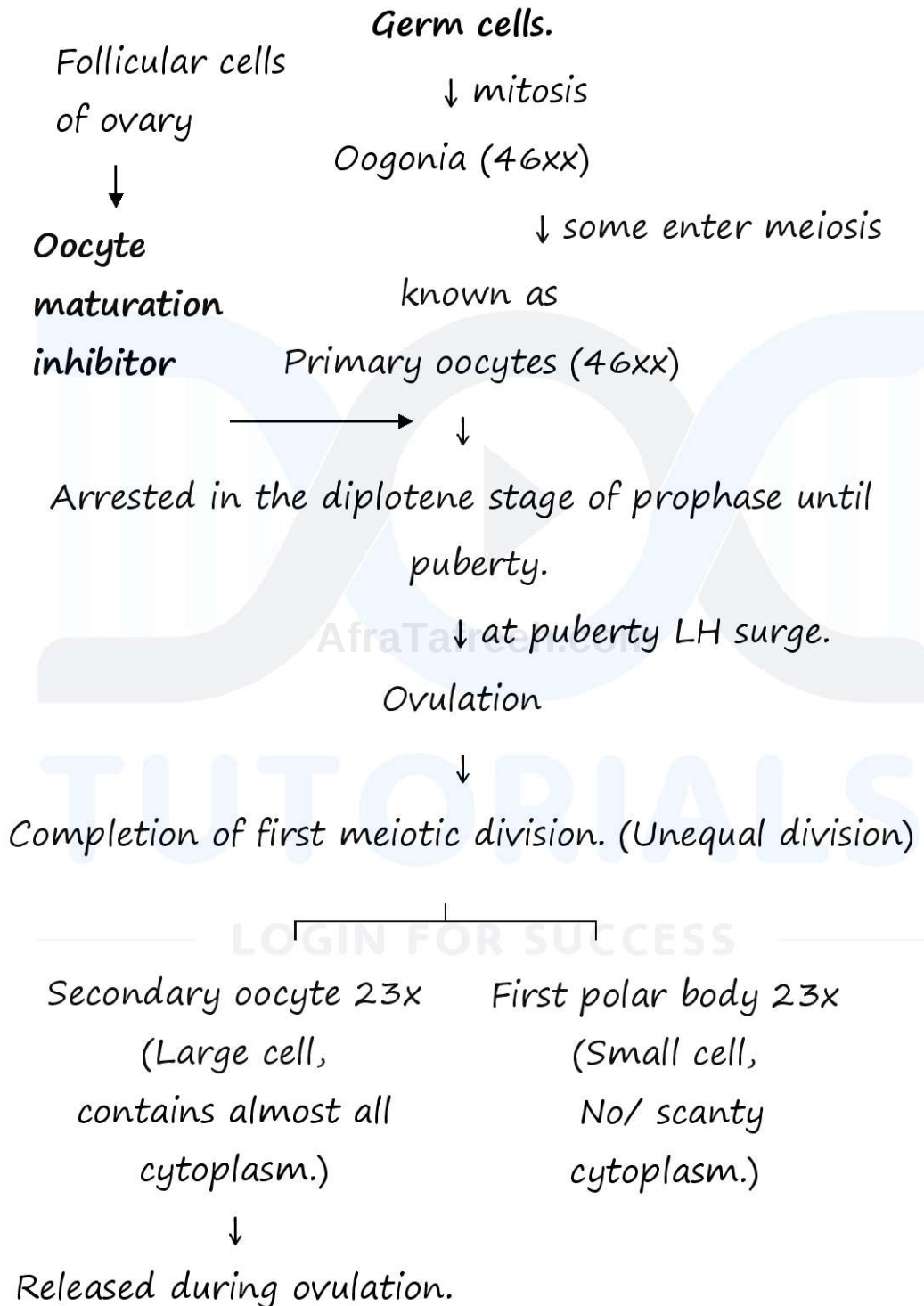
TUTORIALS

LOGIN FOR SUCCESS

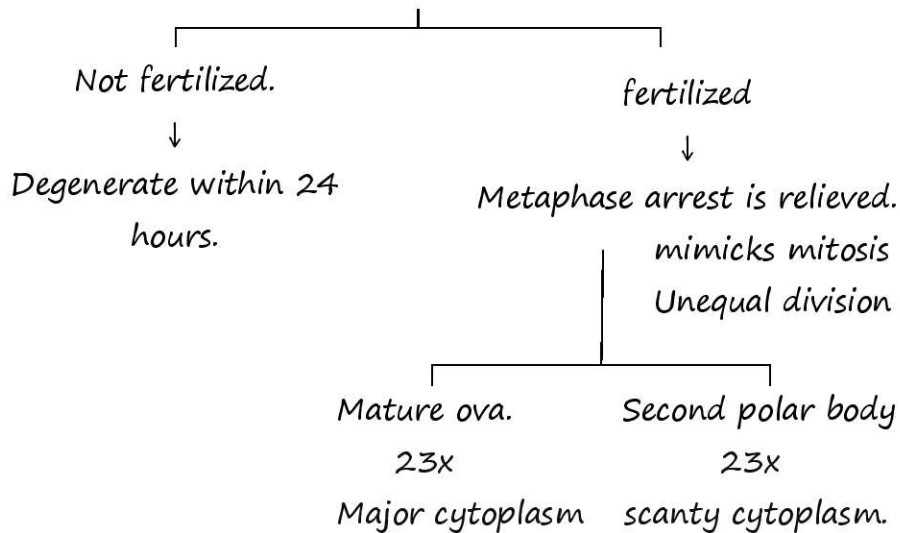
In a newborn ovary → primary oocytes.



Arrested in diplotene stage ^Q of prophase of meiosis I



second meiotic division – arrested at metaphase ^Q



- The 2nd meiotic division is a multiplication division not a reduction division.

^QMaximum number of oocytes – at 20 weeks of IUL.

NUMBER OF OOCYTES

20 weeks of intrauterine life	7 million
At birth	2 million (1-2 million)
Onset of puberty	4,00,000 (4 lakh)
Depletion rate	1000/month
Released During entire reproductive period on ovulation.	400

- Type of oocyte seen ^Q

- In newborn's ovary → primary oocyte
- In reproductive period → primary oocyte
- Newborn → menopause → primary oocyte

^Q Karyotype of:

- Oogonia – 46xx
- Primary oocyte – 46xx
- Secondary oocyte – 23x
- Mature ova – 23x
- First polar body – 23x
- Second polar body – 23x.

Mature ova

- Largest cell in the body

Measures 130 microns in diameter

Karyotype → 23x

It has 2 membranes { Outer → zona pellucida
Inner → vitelline membrane

Space between membranes → perivitelline space.

It contains polar body

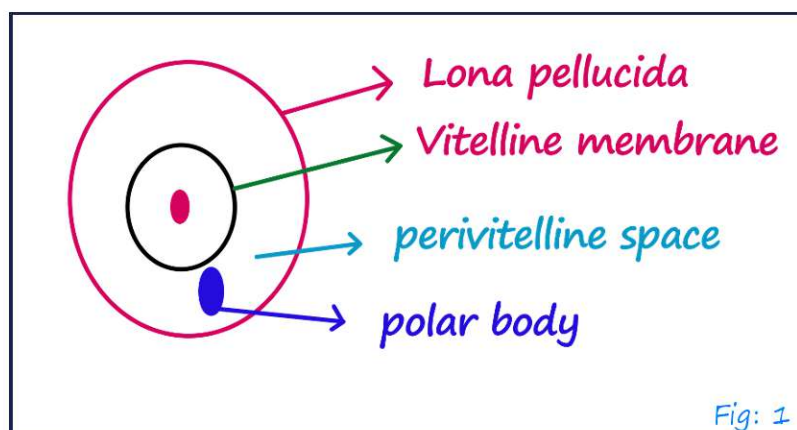


Fig: 1

Perivitelline space

↓ with

One Polar body → secondary oocyte with first polar body

Two Polar bodies → mature ova with first and second polar body.

No polar bodies → primary oocyte

Two polar bodies → indicates that fertilization has occurred.

Spermatogenesis

00:43:54

Definition:

- Process of development of spermatids and their differentiation into mature sperms is spermatogenesis.

Steps of spermatogenesis:

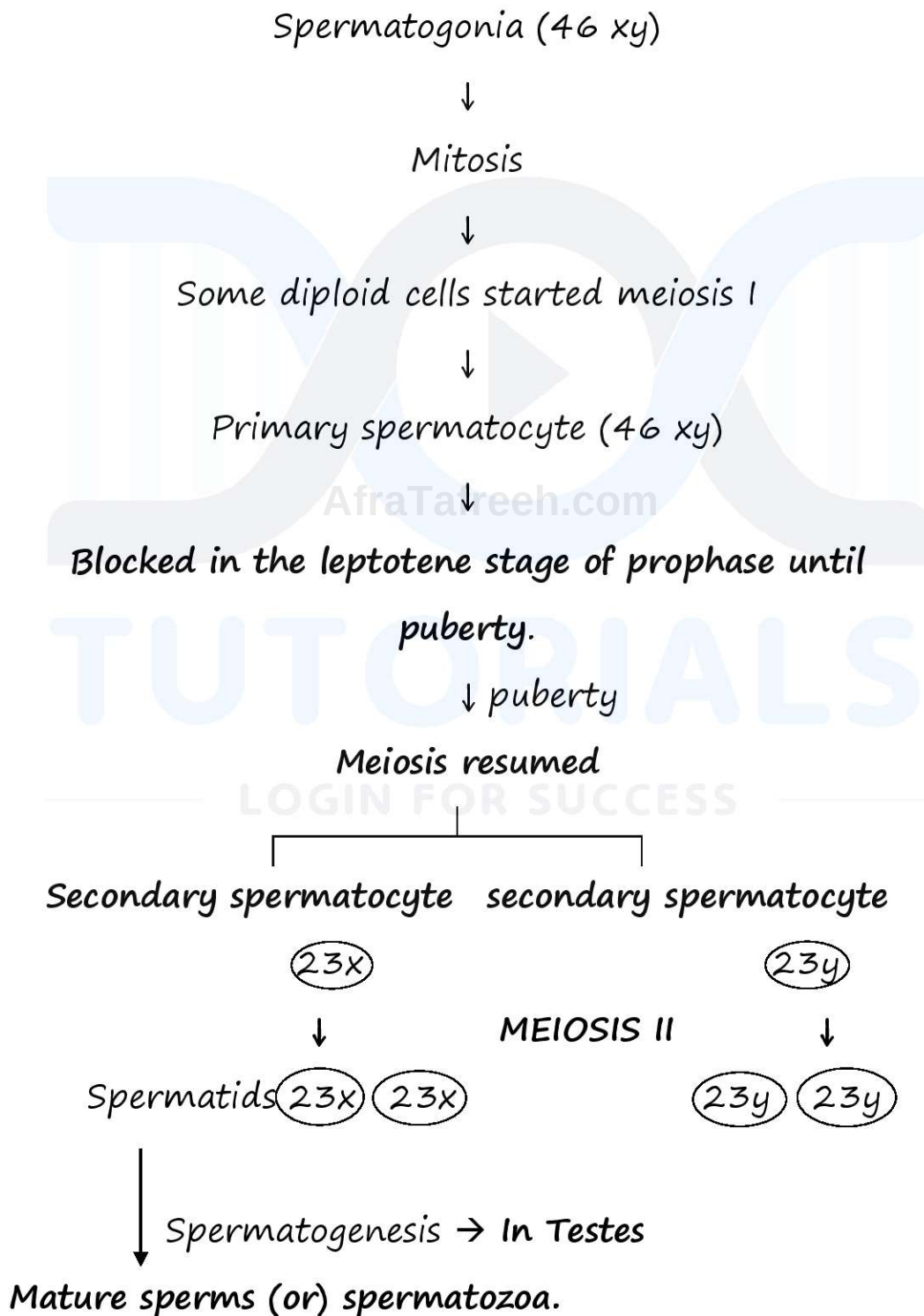
1. Mitosis = spermatocytogenesis = proliferation
 2. Meiosis = reduction
 3. Differentiation = spermiogenesis
 4. Extrusion = spermiation
 5. Maturation → occurs in epididymis.
- } Occurs in testes

Time taken:

- Spermatogenesis (mitosis → extrusion) = 74 days. ^Q

- Spermiogenesis = 14 days.
 - Epididymal maturation = 2 – 14 days
- Primitive germ cell → spermatogonia.

Spermatogenesis:



Changes during spermiogenesis and epididymal maturation

00:57:58

Changes occurring during spermiogenesis:

1. Elongation and condensation of nucleus to form sperm head
2. Acrosome formation – golgi apparatus produce lysosome like granules which form acrosomes.
3. Reorganization of cytoplasm leads to formation of the middle piece and tail.

Middle piece = mitochondria = motility

These spermiogenesis changes are → structural changes,

Take place in → testes.

Changes occurring during epididymal maturation:

- **These are functional changes**
 - **Take place in → epididymis**
1. Increase in negative charge of sperm head.
 - Facilitates attachment to zona pellucida
 2. Activation of acrosomal enzymes ^Q synthesized in testis
 3. Sperm motility ^Q
 - **Sperms attain motility only in epididymis ^Q**
 4. Reabsorption of testicular fluid and concentration of sperms. ^Q

Where are

1. Acrosomal enzymes synthesized → testis
2. Acrosomal enzymes activated → epididymis
3. Acrosomal enzymes released → fallopian tube during fertilization.

Acrosomal enzymes:

- Hyaluronidase
- Neuraminidase like factor
- Cumulus dispensing enzyme
- Pro-acrosin → Acrosin.

Acrosomal reaction:

- The process of release of acrosomal enzymes occurs in the fallopian tube – when sperm come in contact with follicular cells.

Capacitation:

Process of achieving hypermotility by efflux of H^+ and influx of Ca^{+2}

Occurs in the cervix / uterus / tubes.

Most common site for capacitation → cervix ^Q

Sperms
↓ capacitation
Hypermotile
↓

Sperm head binds to zona protein - 2 (ZP - 2)



Activation of plasma membrane



Plasma membrane fuses with outer acrosomal membrane



Acrosomal reaction occurs



Release of acrosomal enzymes



Sperm fuses with ZP -3



Completion of second meiotic division / fertilization



Hardening of zona occur → Zona reaction



To prevent polyspermy.

Important MCQs

1. Number of spermatids developing from 1 spermatogonium → 512
2. Lifespan of a secondary spermatocyte → 1.1 to 1.7 days
3. After epididymal maturation, sperms are stored in → vas deferens
4. Life span of human oocyte → 12-24 hours
5. Life span of human sperm (mature sperm) → 48 - 72 hours

6. Common site of fertilization → ampulla of fallopian tube
7. Longest and widest part of FT → ampulla
8. Liquefaction of semen is due to → enzymes from prostate
9. Normal time taken for liquefaction → 20-30 minutes
10. Sperms that remain in vagina are immobilized in 2 hours
11. Cervix → acts as reservoir of sperm → up to 3 days
12. Vagina → acts as reservoir of sperm → for 2 hours

Fertilization and implantation

01:25:21

Fertilization: fusion of sperm with mature ova –

Time for fertilization	What happens
30 hours	2 cell stages
40-50 hours	4 cell stages
72 hours	12 cell stages
96 hours	16 cell stages (morula)
Day 5	blastocyst

Blastocyst:

Blastomere with fluid is called blastocyst
cells in the blastocyst are called blastomere

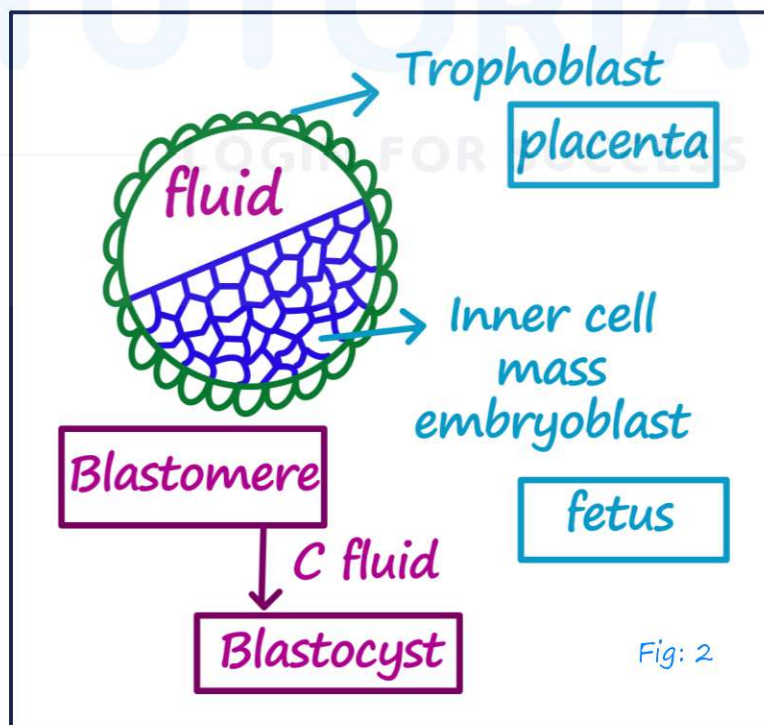
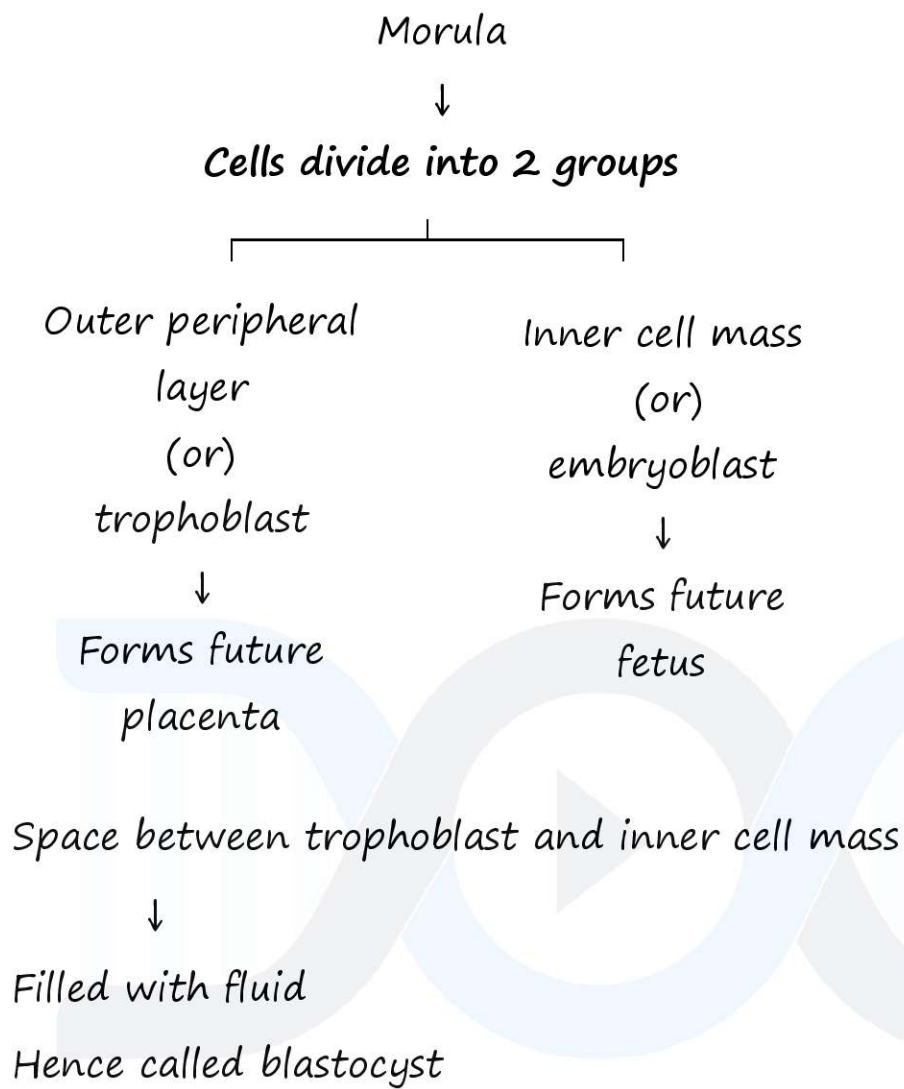
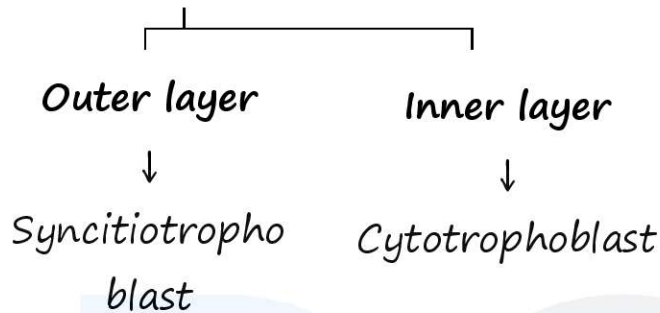


Fig: 2

Zone hatching:

- Shedding of Zona pellucida is called Zona hatching
(occurs at 4th to 5th day of post fertilisation)

Trophoblast:



Implantation

01:31:54

Implantation

- The process by which developing embryo is attached to decidua.
- Also called as nidation ^Q

4 Stages:

- Apposition
- Adhesion
- Penetration
- Invasion

Implantation begins on → D6 post fertilization

(or)

D20 of menstrual cycle

Factors responsible for implantation:

- Selection

- Integrin
- Cadherin
- Matrix metalloproteinase
- Epidermal growth factor
- VEGF
- Troponin
- Heparan sulfate
- Proteoglycan.

Types of implantations in humans → interstitial implantation^Q

Decidua

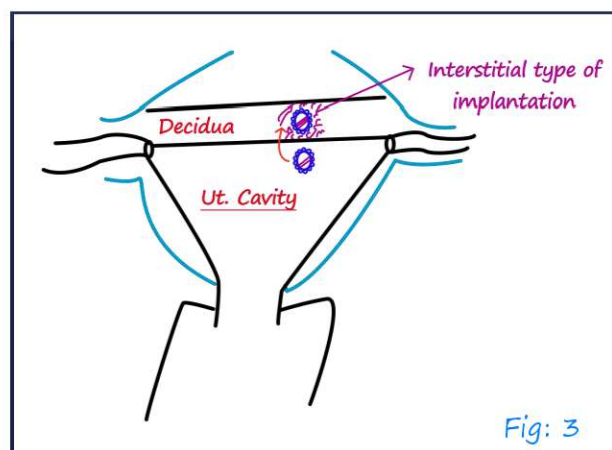
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- Decidua → endometrium of a pregnant uterus
 - It has dilated glands
 - Dilated blood vessels.

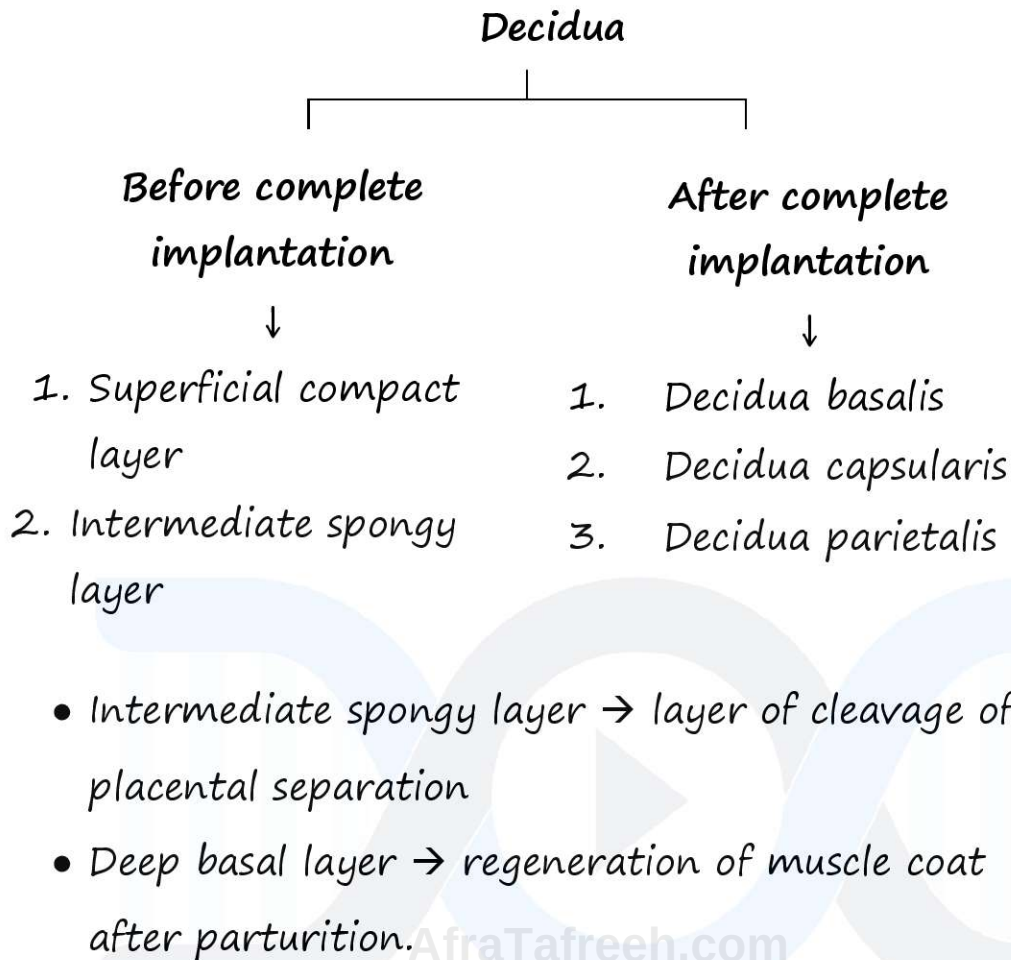
Blastocyst is completely surrounded by decidua on all sides



Interstitial type of implantation.



Decidua



Decidua after complete implantation:

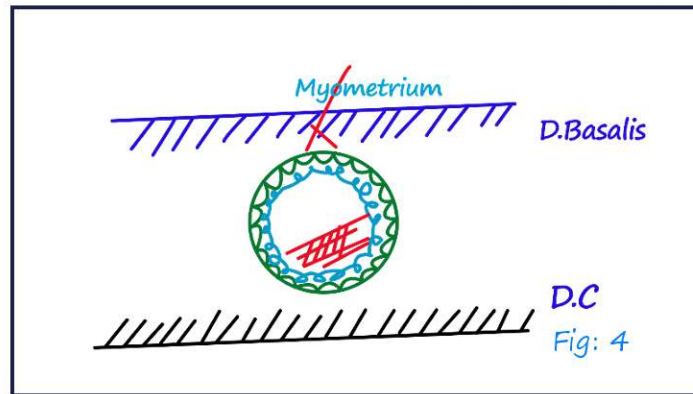
- Decidua basalis / D. Serotina → between blastocyst and myometrium
- Decidua capsularis / D. Reflexa → between blastocyst and uterine cavity
- Decidua parietalis / D. Vera → decidua not involved in the implantation site

When decidua capsularis meets D. parietalis.



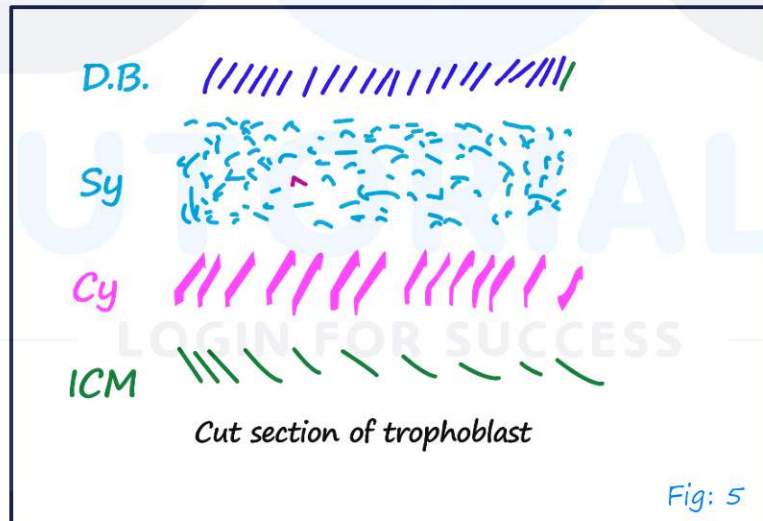
Endometrial cavity gets obliterated

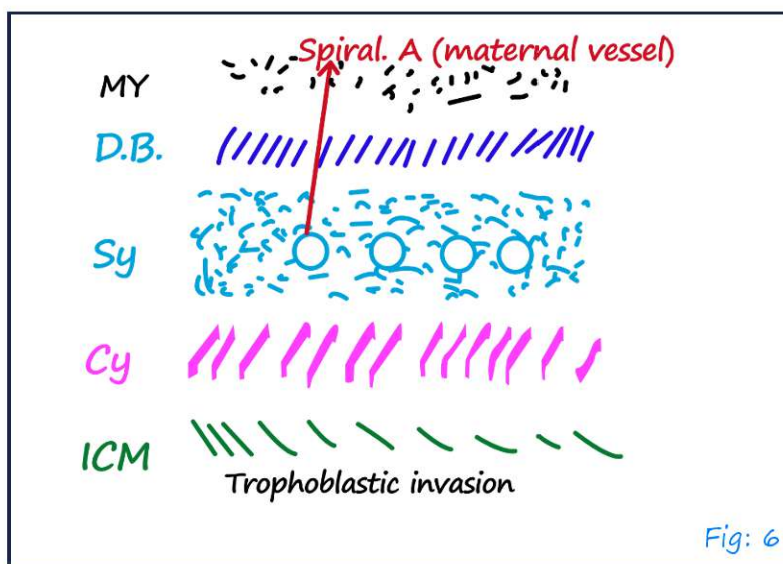
Occurs by: fourth month (or) 16 weeks POG



On cut section

- Myometrium
- D. basalis
- Syncytiotrophoblast
- Cytotrophoblast
- Inner cell mass

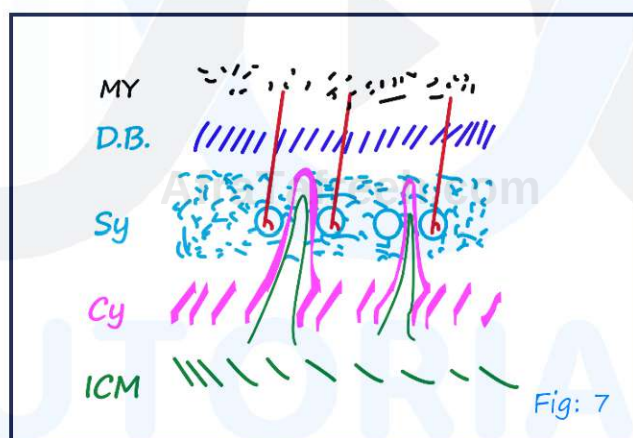




Primary villi – formation



Cytotrophoblast invading syncytiotrophoblast



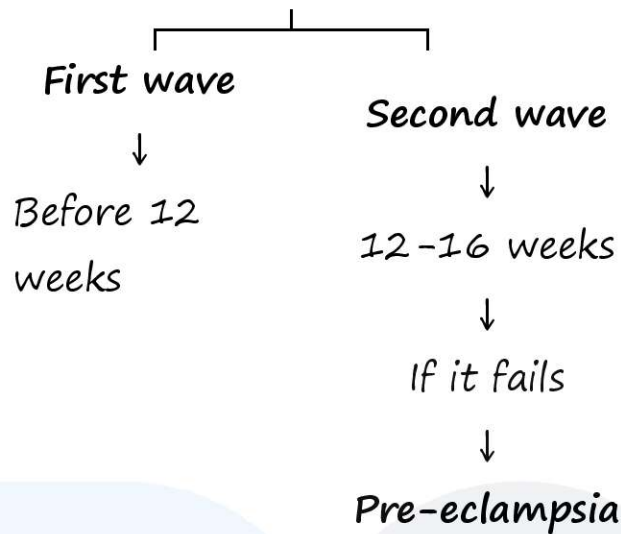
Secondary villi – formation



Inner cell mass invading primary villi

- Tertiary villi → fetal blood vessels into 2° villi.

Trophoblastic invasion



Stages

- 1st wave before 12 weeks
- 2nd wave by 12 - and 16-weeks failure of which leads to preeclampsia
- Primary villi → 1° → syncytiotrophoblast + cytotrophoblast → D13
- Secondary villi → 2° → 1° villi + inner cell mass → D16
- Tertiary villi → 3° → 2° villi + fetal blood vessel. → D21

Uteroplacental circulation

01:59:58

- Uteroplacental circulation
 - The process of maternal blood filling the (Lacunae) (or) placenta – is uteroplacental circulation.
 - Begins on – D11 Q
- Fetoplacental circulation:
 - Begins when fetal heart starts pumping

Begins on – D22 ^Q → Fetal cardiac activity starts.

Chorion frondosum:

Trophoblast close to decidua basalis

↓ grow to form

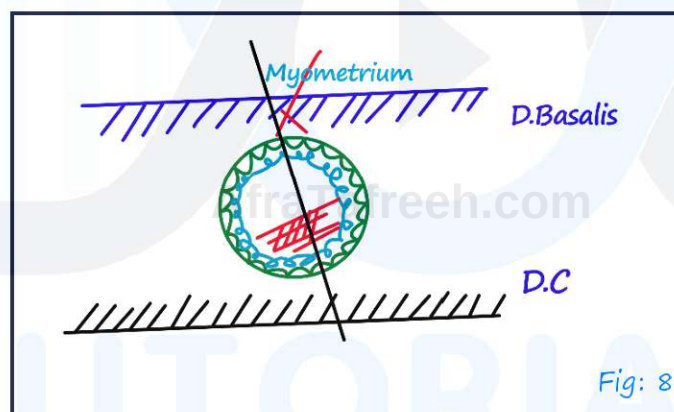
Future placenta

↓

Is known as chorionic frondosum

Chorion laeve:

- Trophoblast → away from D. Basalis. Will degenerate and known as chorion laeve



Nitabuch's membrane:

- Fibrinoid deposit on the syncytiotrophoblast outside the trophoblastic shell.
 - Prevents the migration of placenta deep into decidua.
- If Nitabuch's membrane is absent

↓

Placenta invade decidua/myometrium

↓

Placenta accrete / increta / percreta ^Q

AMNIOTIC FLUID

Functions of Amniotic Fluid

1. Lung development
2. GIT development
3. Space for fetal movements
4. Protects cord compression
5. Bacteriostatic

Volume of Amniotic fluid

- 10 weeks - 30 ml
- 12 weeks - 50 ml
- 16 weeks - 200 ml
- 20 weeks - 400 ml
- 34 weeks - 1000 ml [maximum]
- At term - 800 ml

Physical properties

- PH - >7.2 Alkaline ^Q
- Osmolality - 260 mosm/L (Hypotonic to plasma and Isotonic to fetal urine)
- Specific gravity - 1.010

Source of Amniotic Fluid

00:05:47

-
1. Ultra-filtrate from maternal plasma

Big placenta → increase Volume

Small placenta → decrease Volume

2. Fetal urine

Increase Urine → Volume decrease

Increase Urine → Volume decrease

3. Fetal skin □ keratinizes at 20 weeks of intra uterine life



Decrease Production

- Neural tube defect → increase Liquor
- Big placenta (Placentomegaly) - Multiple pregnancy, Rh Incompatibility,
- diabetes increases liquor formation
- Small placenta → Volume (IUGR, scarring of placenta, in TORCH Infections).

Constitution

- Water - 98%
- Glucose - 20 mg%
- Urea - 30 mg%
- Protein - 0.3mg%
- NPN (Non-Proteinaceous Nitrogen) - 30mg%
- Uric acid - 4mg%
- Creatinine - 2 mg%
- Lipid - 50mg%
- Hormones - Prolactin/Insulin/Renin

Volume Regulation

00:17:35

- Production
 - Fetal urine → 650-1000ml/day
 - Fetal lung secretion → 350ml

	Source
1 st trimester	Maternal plasma/fetal skin
2 nd trimester	Maternal plasma/Fetal skin + Fetal urine
3 rd trimester	Fetal urine

- Resorption
 - Fetal swallowing - 750ml
 - Intramembranous flow - 400ml

Colour

Colour	Seen in
Colour less/transparent	preterm liquor
Milky white & turbid (lanugo/vernix Caseosa)	mature liquor
Green-Meconium staining	fetal distress
Golden yellow	Rh Incompatibility
Saffron	Post maturity
Dark (red) - Blood stained	Abruptio placenta
Brown/Tobacco juice	Intrauterine fetal death

AFI

- Amniotic fluid index → Sum of the largest vertical pockets
- Normal AFI → 5- 25cm

	AFI (cm)	SDP (cm)
Normal	5-25 cm	2-8 cm
Oligohydramnios	<5	<2
Polyhydramnios	>25	>8

Causes for polyhydramnios

00:31:57

Mechanism	Anomaly Examples
Impaired Swallowing (CNS)	Anencephaly Hydranencephaly Holoprosencephaly
Impaired Swallowing (Craniofacial)	Cleft lip/palate Micrognathia
Tracheal compression or obstruction	Neck veno lymphatic abnormality CHAOS
Thoracic etiology (Mediastinal shift)	Diaphragmatic hernia Cystic adenomatoid malfunction

Cardiac Arrhythmia	Tachyarrhythmia: Atrial flutter, atrial fibrillation, Supraventricular tachycardia Bradyarrhythmia: Heart block
GI obstruction	Esophageal atresia Duodenal atresia
Renal Urinary	Ureteropelvic junction obstruction Bartter syndrome
Neurological or Muscular etiology	Arthrogryposis, akinesia sequence Myotonic dystrophy
High output cardiac state	Ebstein anomaly Tetralogy of fallot with absent pulmonary valve Thyrotoxicosis
Functional cardiac etiology	Cardiomyopathy, myocarditis

Complications

- Dyspnoea /orthopnoea (due to over distension)
- Edema
- Oliguria
- Abruptio
- Postpartum Hemorrhage
- Preterm labor

Management

- Amnioreduction (under USG guidance through abdomen)

Oligohydramnios

00:37:48

- AFI < 5cm
- SDP < 2cm

Causes

- PROM → Most common cause
- Postdated pregnancy → 2nd most common cause
- Bilateral Renal agenesis → most common cause for anhydramnios or severe oligohydramnios
- Preeclampsia/Vascular disease
- Utero placental insufficiency
- AR PCKD (Autosomal recessive polycystic kidney disease)
- Multiple dysplastic kidney (MCDK)
- Bladder outlet obstruction
- Mega cystitis / Microcolon intestinal hypoperistalsis syndrome
- Posterior urethral valve
- Urethral atresia/stenosis
- Persistent cloaca
- Sirenomelia
- Drugs – ACE Inhibitors → Cause Renal Anomalies

ARB'S

NSAIDS → decrease Prostaglandins



Decrease Filtration



Oligo hydramnios

Complications

- Still Birth
- IUGR
- Meconium aspiration syndrome
- Increases risk of C section
- Pulmonary hypoplasia
- CAOS – Chronic Abruptio oligohydramnios sequence

Management

- i. Hydration
- ii. Amnio-infusion → Infuse with Normal Saline during intrapartum to resolve cord compression

PLACENTA AND UMBILICAL CORD

Weight of placenta	470 gm (500g)
Shape	Round to oval/discord
Diameter	22 cm
Thickness	2.5cm (2-4cm)
Number of vessels	2 arteries + 1 vein (3 vessel cord) • Right umbilical vein disappears • Left umbilical vein-left back
Maternal surface	Also called Basal plate
Fetal surface	Also called chorionic plate
Length of cord	40-70cm
Short cord	< 32 cm
Long cord	>100cm

- Cord is lined by Wharton's jelly which contains WBC,

Tissue macrophages



Known as Hoffbauer cells ^Q

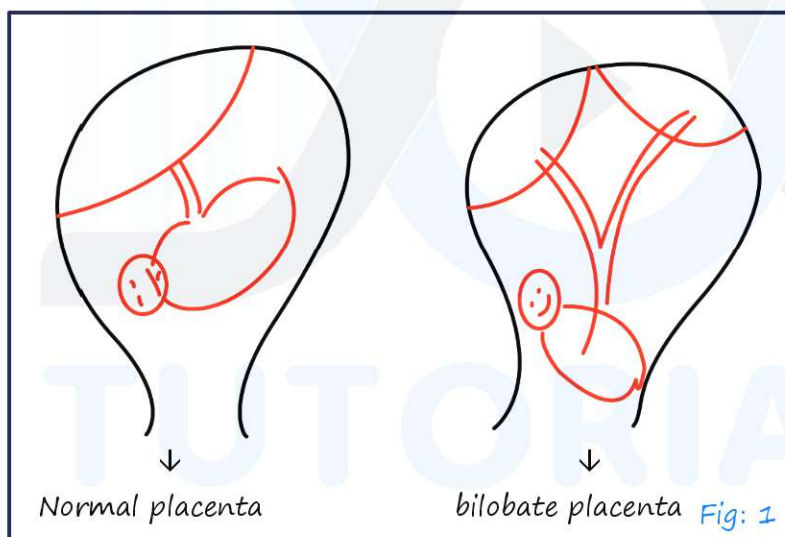
Abnormalities of Placenta

00:03:40

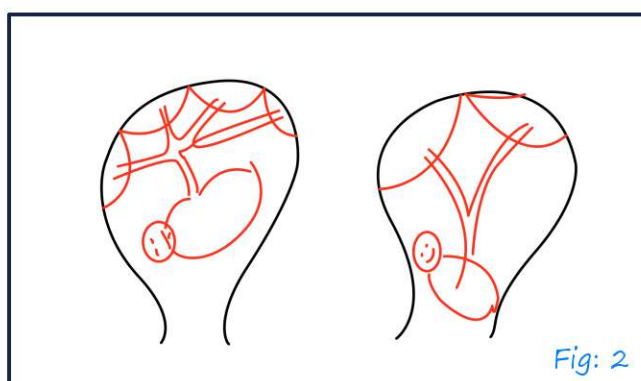
- Bilobate/Bipartite/Placenta duplex
- Multilobate
- Succenturiate

- Membranous placenta
- Ring shaped/Annular
- Fenestrated placenta
- Circummarginate placenta
- Circumvallate placenta
- Battle Dore placenta
- Velamentous insertion
- Furcate placenta

Bilobate/Bipartite/Placenta duplex



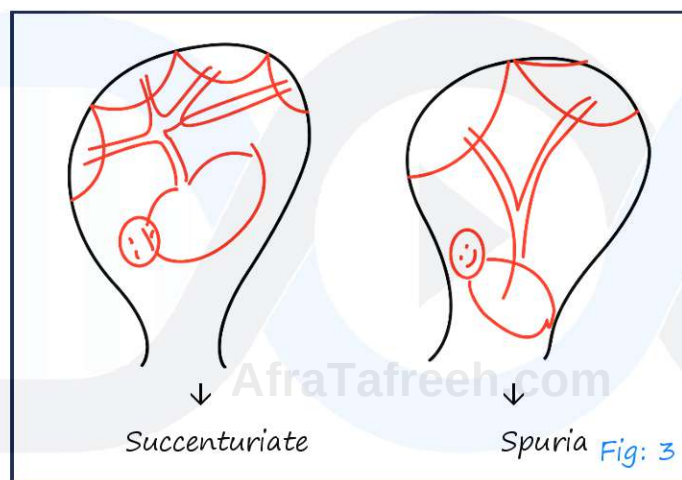
- Both lobe same size
- Multilobate: Placenta has many lobes



- High risk of PPH, sepsis, infection due to retained bits of placenta

Placenta succenturiate & spuria

- One main lobe and lobe & another accessory lobe is present
- Both lobes unequal sizes
- Main lobe is big, accessory lobe is small



- In succenturiate there is communicating blood vessels between two lobes
- In spuria there is no communicating blood vessel between the two lobes
- The umbilical cord gives branch to main lobe
- Accessory lobe gets a branch of communicating vessel or sometimes it may not receive any blood at all

Membranous Placenta

- Placenta or villi covers entire uterus

- When it takes shape of a ring, we call it Ring/Annular placenta

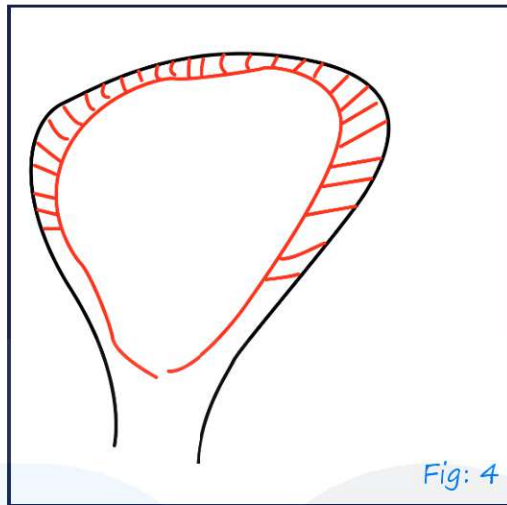


Fig: 4

- PPH can occur

Fenestrated placenta

- One segment or cotyledon is naturally missing
- There's an empty space, lacuna

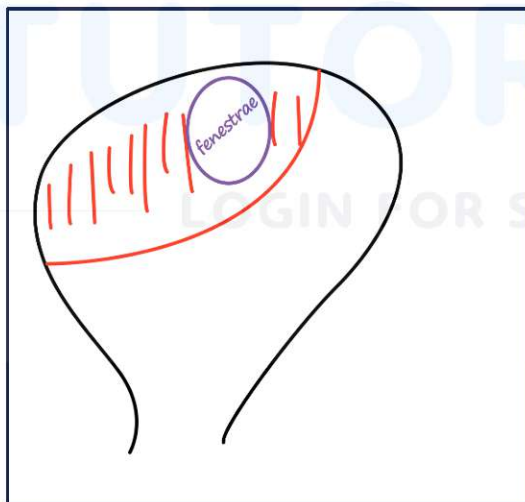
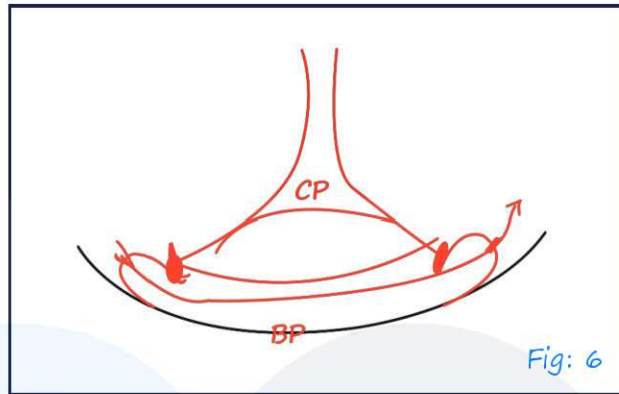


Fig: 5

Circummarginate placenta

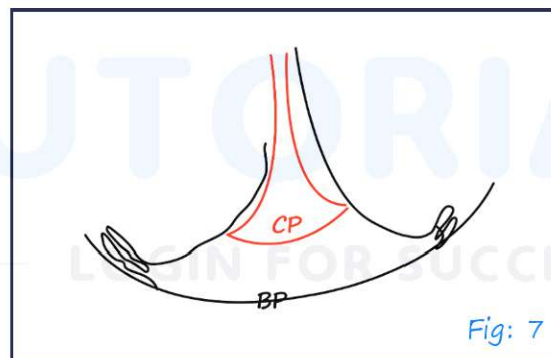
- Usually maternal & foetal plate (basal & chorionic plate) are equal in size

- Chorionic plate < Basal plate circummarginate placenta
- Basal plate appears swollen & fibrins & blood clots project from sides



Circumvallate placenta

- Chorionic plate < Basal plate
- Amniotic membrane forms extra folds which make Basal plate appear bigger

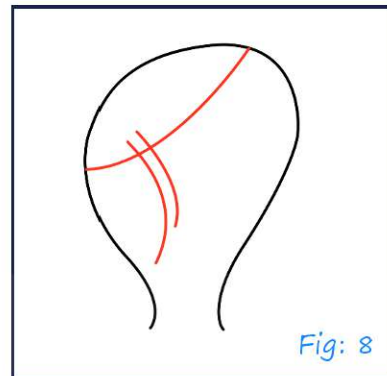


Complications of circumvallate & circummarginate

- APH
- Abruptio
- IUD
- Preterm labour

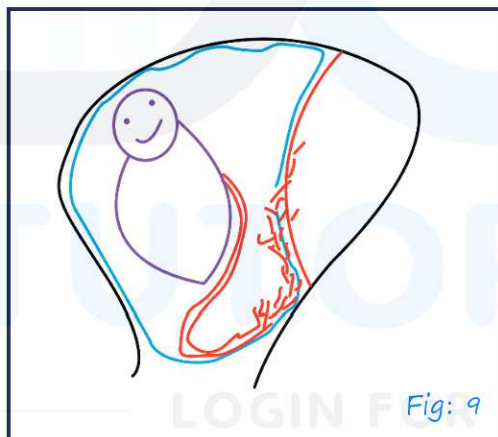
Battle Dore placenta

- Cord is inserted at side instead of centre



Velamentous insertion of cord

- Cord before entering placenta gives numerous branches which run on the amniotic membrane

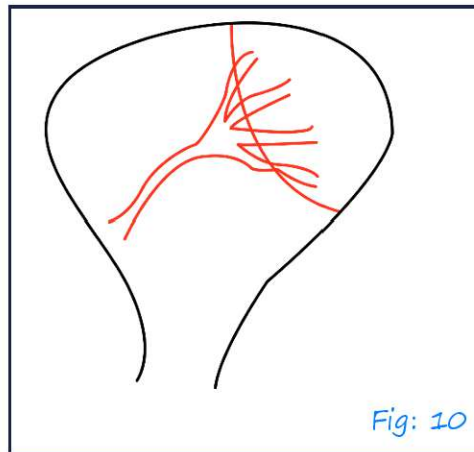


Hazard

- Fetal vessels are left unsupported & when vaginal examination done, the finger might injure vessels & damage & bleeds – this is Vasa Previa

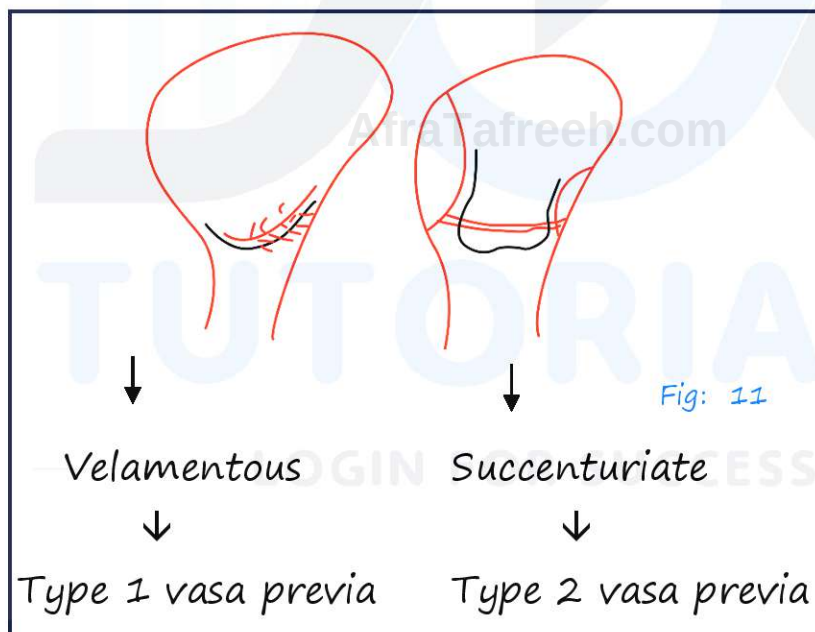
Furcate placenta

- Cord divides into branches before entering placenta but do not run-on amniotic membrane



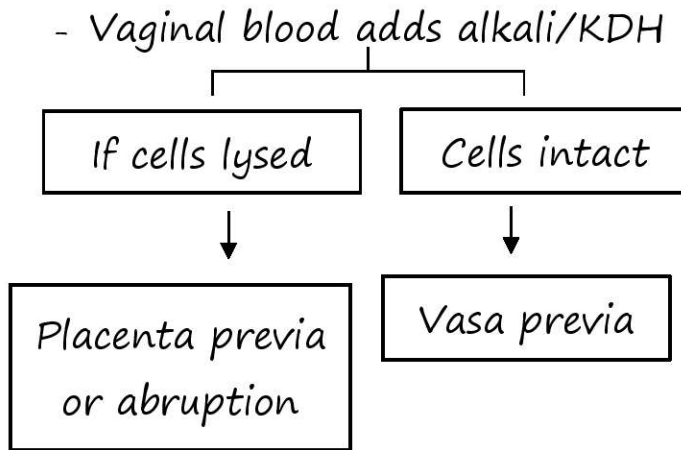
Vasa Previa

- Due to
 - Velamentous insertion
 - Succenturiate



Hazard

- Fetal haemorrhage
- IOC → TVS with color Doppler ^Q
- If patient is severely bleeding – singers alkali
Denaturation test or APT test
- Fetal RCB is resistant to acid/alkali



Causes of placentomegaly (Placenta thickness > 4cm)

00:26:38

- Diabetes
- Anemia
- Hydrops fetalis
- Infection (TORCH/Parvo virus)
- Syphilis
- Partial mole
- Placental mesenchymal dysplasia
- Chorioangioma

LOGIN FOR SUCCESS

Amnion nodosum

- Numerous small light tan nodules on amniotic membrane
- Contains fibrin and foetal squames
- Complication: oligohydramnios

Amniotic Band Sequence

- Anatomical disruption of uterine cavity by bands made of Amniotic membrane

Amniotic bands tether together



Leads to limited foetal movements



Which leads to limb reduction

- Facial cleft
- Omphalocele

Breus mole ^Q

- Massive subchorionic hematoma

Funisitis ^Q

- Inflammation of chorionic plate and umbilical cord.
- Umbilical cord presenting in labour

Hyrtl anastomosis ^Q

- Connection between the two umbilical arteries usually near the placenta

Single umbilical artery

- Incidence – 0.4 to 0.6%
- Due to increase maternal age tobacco use
- 80% are isolated (not associated with anomalies)
- Most common anomaly CVS anomaly (7%) – VSD

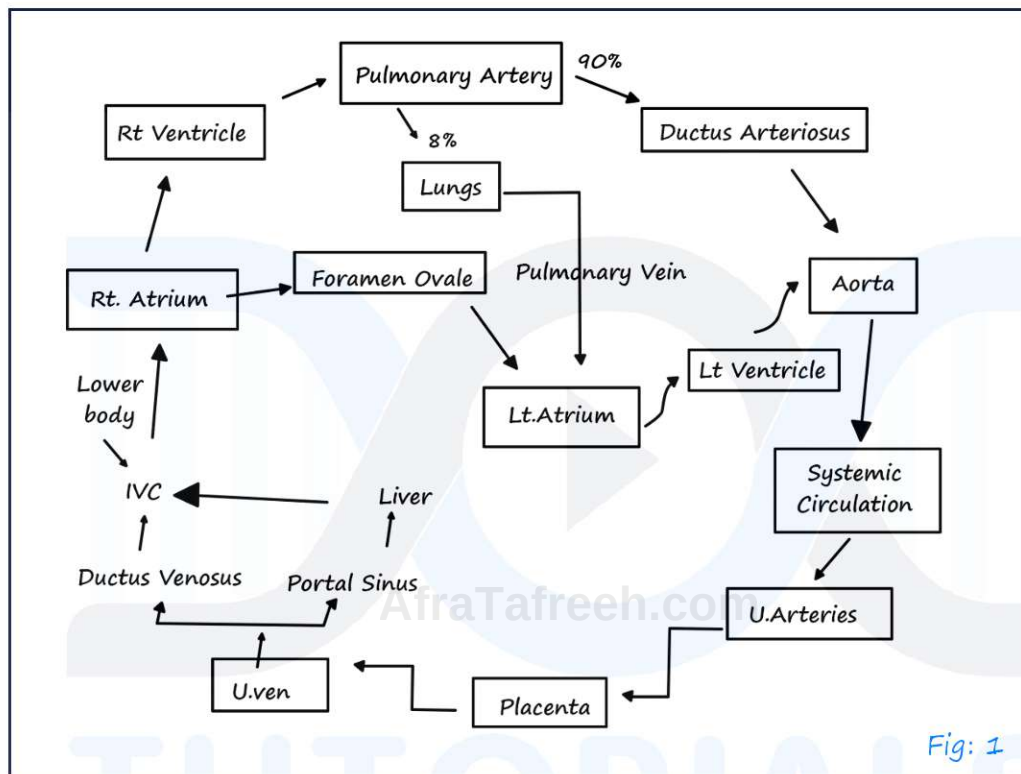
- Renal anomaly (5%) – renal pelvic dilation
- Associated with Aneuploid – 4%^Q
 - ↓
 - Most common: Edwards^Q
 - Trisomies
 - Triploidy
- Can lead to IUGR or Oligohydramnios



FETAL CIRCULATION

Schema

- Foetal circulation begins with Placenta and ends with it



- 90% of blood from Pulmonary Artery enter Ductus Arteriosus and then into descending Aorta
- 8% of the blood from Pulm. Artery goes to lungs just for nourishment
- Oxygen saturation in Umbilical Vein is 80%
- Cardiac output for foetus is 350 ml / Kg / minutes
- Cardiac output for new born is 500ml / minutes

Changes at birth

- Closure of

Umbilical Arteries	immediate functional closure
	Obliteration takes 2 – 3 months
Umbilical Veins	
Ductus Venosus	
Ductus Arteriosus	immediate functional closure
	Obliteration takes 1– 3 months
Foramen ovale	Anatomical closure / complete
	Obliteration takes 1 year

- fall in IVC & right atrial pressure

Ligaments: (vessels form ligaments)

Distal Umbilical Artery	→ lateral umbilical ligament
Proximal umbilical artery	→ superior vesical artery
Umbilical vein	→ ligamentum teres
Ductus Venosus	→ ligamentum venosum
Ductus arteriosus	→ ligamentum arteriosum

- Cardiac output of New-born is 500ml /min

HEMATOLOGICAL CHANGES IN PREGNANCY

- Blood volume increases by 40-45% in pregnancy
- It starts increasing by 6 weeks → 1st trimester
- Blood volume shows maximum change in 2nd trimester
- Peaks in 3rd trimester → 32-34 weeks
 - Plasma volume increased by 40-50%
- RBC mass increases only by 20% (net hemodilution – 450ml)
- Haematocrit drops and during pregnancy
 - Non pregnancy (38%)
 - Pregnancy (32%)
- Haemoglobin concentration decreases
- Viscosity decreases with pregnancy

Iron metabolism

- Daily iron requirement of non-pregnant lady -3-4 mg/d
- Daily iron requirement of pregnant lady -6-7 mg/d

Iron requirement during pregnancy

- To expand RBC: - 500mg of iron
- Uterus, fetus and placenta-300 mg
- Daily loss: - 200mg of iron
- Blood loss during delivery = 200mg
- Amenorrhea during pregnancy saves 200 mg

- 1000 mg is the Net requirement iron

Markers such as: -

- Serum iron – decreases
- Serum ferritin – decreases
- Hepcidin – decreases
- Transferrin – increases
- TIBC – increases
- Percentage saturation (Ferritin/TIBC) = decreases with pregnancy

Immunological functions.

00:12:50

Pregnancy -Immunocompromised state

- Leukocytosis → WBC increases upto 15,000
- In puerperium → increases upto 25,000
- B lymphocyte – normal
- T lymphocyte → TH₁ – Pro inflammatory → (IL₂ IFN α , TNF) decreases/down regulated
→ TH₂ – Anti-inflammatory
↓
Upregulated (IL₄, IL₁₀, IL₁₃)
- Neutrophils – Increases in pregnancy (Neutrophilic leucocytosis)

Inflammatory markers

- | | | |
|--|---|--------------------------------|
| <ul style="list-style-type: none"> • ESR • CRP | } | <p>Increase with pregnancy</p> |
|--|---|--------------------------------|

- Complements C_3C_4
- Leukocyte alkaline phosphatase
- Procalcitonin
- Increase ESR is due to increase in fibrinogen levels & Serum globulin levels
- Cell mediated immunity is compromised
- Humoral immunity takes the upper hand during pregnancy

Clotting system

- Pregnancy is pro-coagulation state
- Clotting factor increase with pregnancy
- Fibrinolytic system is depressed
- Anti-thrombotic proteins decrease with pregnancy
- Pregnant lady is at high risk for DVT usually seen in left side of the body
- Clotting factor which mainly increase with pregnancy is
 - Clotting factor, I – fibrinogen
 - i. In non-pregnant state 200-400 mg/dl (500 mg%).
 - ii. In pregnant: - 300-600 mg/dl (450mg%)
 - iii. So, it increases by 50%

Clotting factor which decreases in pregnancy are: -

- Factor 11,13
- All other factor increases in pregnancy

- In pregnancy, the conversion of plasminogen to plasmin is decreased so plasminogen level increases
- Fibrin stabilizing factor also drops with pregnancy so
- Fibrinolytic activity decreases
- Protein C]
• Protein S] Decreases
- Anti-thrombin
- Platelet count drops with pregnancy
- Increase consumption of platelet & Hypersplenism
- BT and CT → Unchanged during pregnancy

TUTORIALS

LOGIN FOR SUCCESS

CARDIOVASCULAR CHANGES OF PREGNANCY

- Heart rate increases by 10 beats/min.
- Pulse rate increases by 10 beats/min.
- Progesterone is a smooth muscle relaxant. So, as progesterone increases during pregnancy peripheral resistance decreases and pulmonary vascular resistance decreases
- Stroke volume increases as blood volume increases
- End diastolic volume increases
- Cardiac output also increases ($HR \times SV$)
- Ejection fraction (SV/EDV) remains unchanged

Cardiac Output (CO)

- Begins to increase by 5 weeks
- Maximum during pregnancy at 32 weeks.
- CO increases by 40% during pregnancy
- CO increases by 50% during labour.
- CO increases by 70% in the immediate postpartum period because blood from placenta returns to maternal circulation (3RD STAGE OF LABOUR)
- In the 4th stage CO decreases from 70% to 40% (pre labour value).
- CO goes back to pre-pregnant values in about 2 weeks after delivery

Blood pressure

00:08:25

- Both systolic and diastolic blood pressure fall during pregnancy but diastolic pressure falls more than systolic pressure.
- Fall in blood pressure begins by 6 weeks.
- Fall is maximum by 24-26 weeks (2nd trimester)

Apex beat

- Normal position of apex beat is 5th intercostal space medial to mid clavicular line
- In pregnancy apex beat shifts to 4th intercostal space lateral to mid clavicular line
- As all 4 chambers widen during pregnancy the cardiac silhouette enlarges
- Minimal pericardial effusion is present during pregnancy
- In ECG, left axis deviation and inverted T waves may be seen

Heart sounds

- $S_1 \rightarrow$ loud and splitting
- $S_2 \rightarrow$ unchanged
- $S_3 \rightarrow$ loud

Murmurs

- Systolic murmurs of grade 1 (or) 2 are physiological

- These murmurs heard are intensified during inspiration/expiration
- Diastolic murmurs can be present physiologically in 20% of women.

Pressure changes

- Pulmonary capillary wedge pressure
 - Central venous pressure
 - Antecubital venous pressure
- } Unchanged
- Femoral venous pressure increases due to venous pooling in lower limbs (Physiological edema)
 - Femoral venous pressure in non-pregnant lady is 8mmHg and in pregnant lady is 24mmHg.
 - Renin and Angiotensin increase in pregnancy.
 - ANP and BNP remain normal and increase only in preeclampsia
 - PGE_2 increases
 - PGI_2 increases (vasodilator)
 - TXA_2 is a vasoconstrictor
 - If the ratio of PGI_2 to TXA_2 drops it indicates risk of preeclampsia.

PHYSIOLOGICAL CHANGES OF PREGNANCY

Reproductive tract

	Non pregnant Uterus	Pregnant Uterus
Wt.	70g	1 to 1.1 Kg (1000g)
Volume	10ml	5Lit

- Increase in uterus weight is due to Hypertrophy ^Q
- Not hyperplasia

Growth of uterus

- Estrogen & progesterone in → 1st trimester
- Stretching due to fetus → 2nd trimester

Shape of uterus

- Before 12weeks – Piriform
- After 12weeks – Spherical
- Dextrorotation of uterus → Rectosigmoid on left side

Cervix

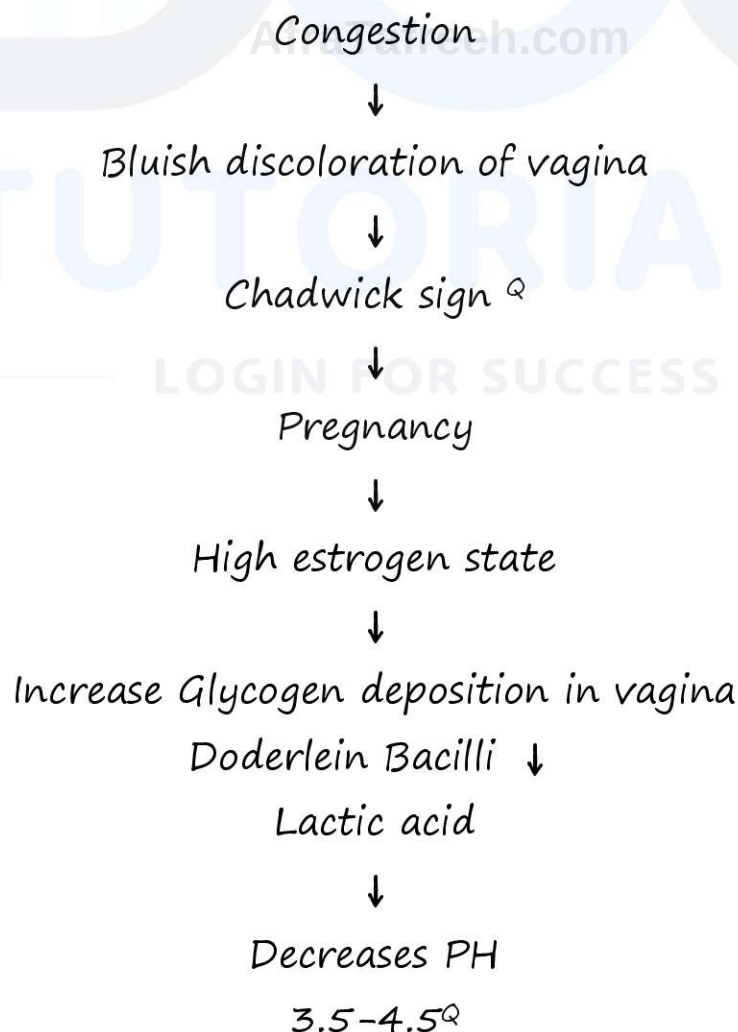
00:05:34

- Softening of Cervix – **Goodell's sign** – 6 weeks
- Glands → Hypertrophy and Hyperplasia
- Increases vascularity, Decreases venous return → Bluish colouration

- Eversion – (Opening up of cervix under influence of estrogen) → Cervix appears – Red, velvety, Bleeds on touch
- Increases mucus production Increases Ferning (due to salt & water content) and Increases estrogen

Vaginal

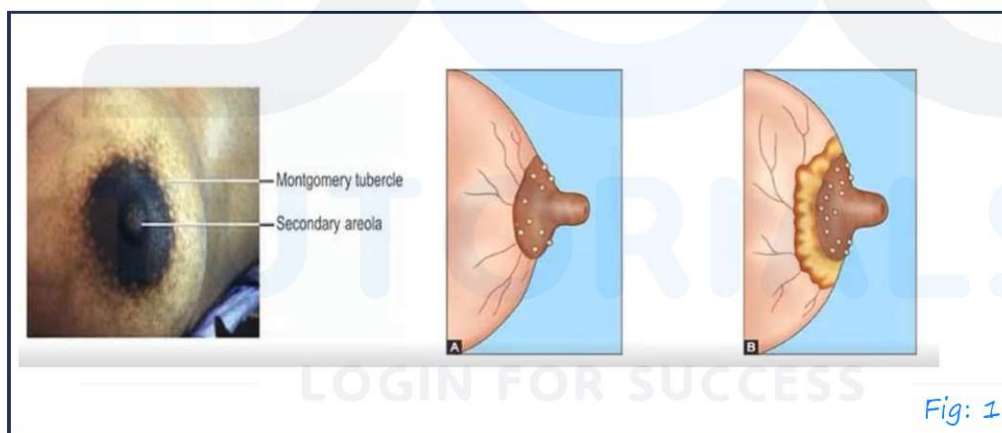
- Normal PH → 4.5 – 5.5
- In pregnancy decrease PH → 3.5 – 4.5^Q
- Increase Vascularity
- Decrease Venous return



- Increase Chances of candidiasis in pregnancy ^Q

Breast

- Estrogen → Ducts
- Progesterone → Alveoli
- Breast tenderer (+)
- Areolar space – Enlarged nipples → Secondary areola
- Colostrum
 - i. Appears by – 12 weeks
 - ii. Thickens by – 16 weeks
- Sebaceous glands – Enlarge – Montgomery's tubercles



- Gigantomastia → Abnormally enlarged breast during pregnancy

Skin

- *Striae gravidarum* – stretch marks (Break down of collagen)



- *Linea Nigra* → Pubic symphysis to xiphisternum



- Increase MSH (Melanocyte Stimulating Hormone)
- *Chloasma* → Mask of pregnancy (In malar eminence)



Fig: 4

- Spider/Nevus/Telangiectasia /Angioma due to Increases E_2
- Diastasis recti

Metabolic changes in Pregnancy

00:20:15

-
- Basal metabolic rate $\rightarrow$ BMR increases by 20% (singleton) and 30% (Twin)
 - Additional Calories
 - 1st trimester 85 Kcal/d
 - 2nd trimester 285 Kcal/d
 - 3rd trimester 475 Kcal/d

Weight gain

- Normal Average wt. gain in pregnancy $\rightarrow$ 12.5kg
 - Fetus – 3-3.5kg ^Q
 - Uterus – 1Kg ^Q
 - Blood – 1.5Kg
 - ECF – 1.5Kg

- Fat – 3.5kg (3.3-3.7kg) ^Q Maximum contribution
- Breast – 0.4Kg
- Placenta – 0.6Kg
- Liquor – 0.8kg

Water

- Extra water – 6.5L
 - 3.5 lit (Fetus/Placenta/Liquor)
 - 3lit (Maternal blood)
- Osmolality → by 10mosm/kg

Protein metabolism during pregnancy 00:26:04

- Gain – 600-1000g
- 500g – fetus/placenta
- 500g – Mother (uterus/breast)
- Daily Requirement of protein – 1.2g/kg/d in 1st trimester
 - 1.5/Kg/d in 2nd & 3rd trimester

Carbohydrate metabolism during pregnancy

- Fasting hypoglycemia
- Postprandial hyperglycemia
- Hyperinsulinemia
- Insulin sensitivity decreases by 70%
- Hepatic gluconeogenesis
 - Hyperglycemia state

Fat Metabolism

- Lipolytic
 - Increase LDL/increase HDL/increase VLDL
 - Lipoproteins increases
 - Apolipoproteins increases
- } **Increases**

Non-Ionized Ca^{+2}
(Calcium bound to albumin)

In pregnancy



Non ionized ca^{+2} decrease
Albumin conc decrease

Electrolyte

- increase GFR
 - Filtration increase
 - Re-absorption increase
- } **Excretion unchanged**
-
- Na^{+} Conc decrease
 - K^{+} Conc decrease
 - Total Ca^{+2} decrease
 - Ionized – normal
 - Non-Ionized – decrease
 - Albumin concentration decrease
 - Mg^{+2} decrease
 - Phosphate – (Normal)
- } **Due to plasma expansion**

Hematology

00:36:02

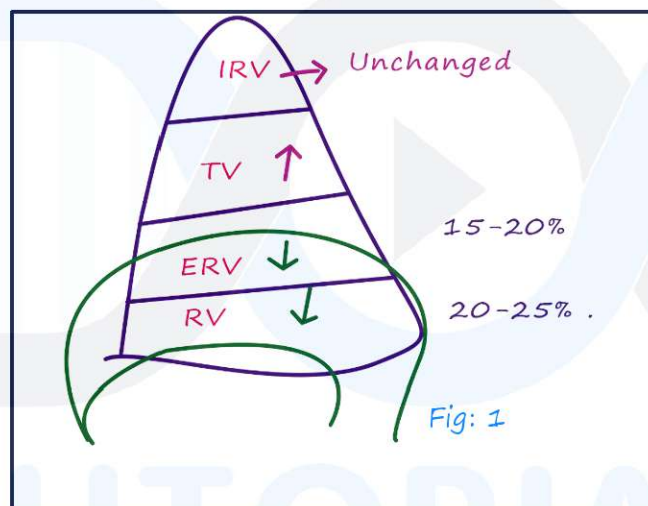
- Blood volume
 - Starts Increasing by 6 weeks of pregnancy
 - Reaches peak 32 – 34 weeks (3rd trimester)
 - Rate of increase is max in 2nd trimester
 - Blood volume increase by 40-45%
- Plasma volume increase by 40-50%
- RBC mass increase by 20%



RESPIRATORY, URINARY SYSTEM AND GI CHANGES IN PREGNANCY

Respiratory System Changes

- Diaphragm rises by 4cm
- Transverse diameter of thorax increases by 2cm
- Circumference of chest increases by 6cm
- Respiratory rate remains unchanged



- Residual volume (RV) decreases by 20-25%
- Expiratory reserve volume decreases by 15-20%
- Tidal volume increases
- Inspiratory reserve volume remains unchanged

Capacities

- Inspiratory capacity = $IRV + TV$ = increases (by 5-10%)
- Functional residual capacity = $ERV + RV$ = decreases (by 20-30%)

- Total lung capacity = IRV +TV + ERV +RV =decreases (< 5%)
- Vital capacity = IRV +TV +ERV = unchanged
- Maximum breathing capacity → unchanged
- Airway conductance increases
- Peak expiratory flow rate increases
- Lung compliance remains unchanged
- Pulmonary resistance decreases
- Maternal AV oxygen difference decreases
- Partial pressure of CO₂ decreases by 10mmHg
- O₂ consumption increases by 20% during pregnancy
- O₂ consumption increases by 40-60% during labour

Urinary System Changes. 00:12:30

- Kidney size increases by 1cm
- Renal plasma flow increases by 80%
- GFR increases by 50%
- Clearance rate increases
- Serum creatinine decreases (0.5-0.7)
- Serum bicarbonate decreases by 4-5mEq/L because minute ventilation increases
- Pregnancy is a state of respiratory alkalosis
- Serum osmolality decreases by 10mosm/kg
- Excretion of soluble vitamins, amino acids and glucose increases
- Pregnancy is a condition with physiological glycosuria

- Ureters are laterally displaced and show hydroureteronephrosis which is more prominent on right side due to dextrorotation

Bladder Changes

- Wall thickness increases
- Increased vessel tortuosity
- Increased bladder pressure leading to urinary incontinence
- Increased urethral length

G.I System Changes

- Gastric emptying time – unchanged
- Relaxation of LES increases risk of GERD

Liver and Gallbladder Changes

- Placental alkaline phosphatase increases
- ALT, AST, GGT, bilirubin decrease
- Synthesis of albumin and globulin increases but concentration decreases due to haemodilution
- Fall in albumin concentration is relatively more
- Leucine aminopeptidase increases, it has oxytocinase and vasopressinase activity because of which there is transient diabetes insipidus in pregnancy
- Gall bladder contractility decreases
- Cholesterol gallstones increase.

FETAL ENDOCRINOLOGY

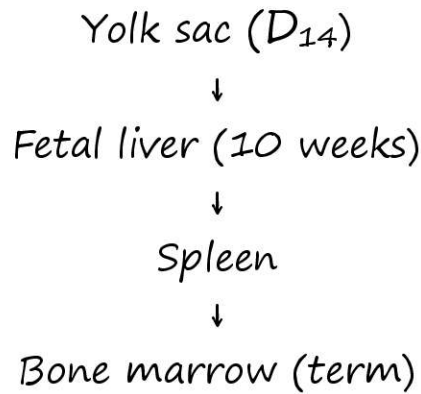
- G – Glucagon → begins by 8 weeks.
 - A – Anterior pituitary hormones – 10 weeks.
 - T – thyroxine – 11 weeks.
 - I – Insulin
 - P – posterior pituitary hormones.
- } 12 weeks

At 12 weeks

- i. Placental sign → cyclical bleeding even after conception until decidua is obliterated → 12 weeks.
- ii. IgG crosses placenta → 12 weeks.
- iii. Urine production in fetus → 12 weeks
- iv. Swallowing → 10 – 12 weeks
- v. Irregular breathing → 11 weeks, regular → 20 weeks.
- vi. Insulin/post pituitary hormones.

At 20 weeks

- i. Sebaceous glands
 - ii. Meconium
 - iii. Regular breathing movements
 - iv. Keratinisation of the skin
- In foetal life, haematopoiesis begins at day 14 after conception and happens in the liver.



- At term, the major site of haematopoiesis in bone marrow.
- In yolk sac, it is Gower 1, Gower 2 and Portland type of haemoglobin.
- In the liver, $\rightarrow$ HbF (α_2, γ_2)
- In bone marrow $\rightarrow$ (α_2, β_2)
- In the early pregnancy, RBC's are megaloblastic/normoblastic.
- Upto 24 weeks, $\rightarrow$ HbF
- From 24 weeks onwards $\rightarrow$ HbF & HbA
- At term, 75-80% $\rightarrow$ HbF type.
- Lifespan of foetal RBC is 80 days and at term becomes 90 days.
- Blood group antigens appear after 38 days (1st trimester)
- Fetal blood has more affinity for O_2 & less affinity to 2,3 DPG
- Resistant to acid/alkali denaturation
- All the HbF is replaced by adult Hb at 6-12 month of life. (After delivery)
- Cord blood levels of Iron, Ferritin, B_{12} & Folate are higher than in maternal plasma.

PLACENTAL HORMONES

Hypothalamic Hormones

- Cell of origin
 - CRH – Syncytiotrophoblast & Cytotrophoblast
 - GnRH – Syncytiotrophoblast & Cytotrophoblast
 - TRH – Syncytiotrophoblast & Cytotrophoblast
 - GHRH – Syncytiotrophoblast & Cytotrophoblast

Pituitary Hormones

- Cell of origin
 - ACTH – Syncytiotrophoblast
 - HCG – Syncytiotrophoblast
 - HCT – Syncytiotrophoblast
 - HPL – Syncytiotrophoblast
 - GH – Syncytiotrophoblast
 - HCG & HPL – Before 5 weeks of intrauterine life
also come from Cytotrophoblast & after, 5 weeks
exclusively from Syncytiotrophoblast

Growth Factors

- Inhibin, Activin – Syncytiotrophoblast
- TGF – beta – Syncytiotrophoblast
- IGF 1,2 – Syncytiotrophoblast
- Epidermal growth factor – Syncytiotrophoblast

Steroids

- Estrogen – Corpus luteum
 - Progesterone – Corpus luteum
 - Cortisol – Decidua/ adrenal. gland of fetus
-] up to 8 weeks then
by placenta

HCG.

00:02:40

- Marker of pregnancy
- Also called as “LH surrogate” because it mimics the action of LH (luteinizing hormone)
 - Biochemical Nature: Glycoprotein
 - Act via: LH receptors
 - Molecular weight: 36,000 to 40,000 Dalton
 - T $\frac{1}{2}$: 36 hours
 - Doubling Time: 48 hours / 2 days (1.4 – 2 days)
 - Subunits: alpha & beta subunit → specific
 - Structurally similar to HCG: FSH, LH, TSH same alpha chain

Source of HCG

- Before 5 weeks: Syncytiotrophoblast & Cytotrophoblast
- After 5 weeks: Syncytiotrophoblast
- Chromosome coding alpha – chain: chromosome 6
- Chromosome coding beta chain: Chromosome 19
- Maximum levels: 50,000 to 1,00,000 MIU/ml

- Earliest detectable: 7 to 9 days after LH surge (day of implantation)
 - Peaks at – 8 to 10 weeks of pregnancy (60-80 days of pregnancy)
 - HCG in liquor = HCG in maternal plasma.
- Placental GnRH stimulate HCG production
- Clearance
 - a. 70 % liver
 - b. 30% kidney

Functions of HCG

- i. Rescue and maintenance of – Corpus luteum
- ii. Stimulates – Leydig cell replication (Leydig cell → testosterone)
- iii. increase testosterone secretion → Male sexual differentiation
- iv. Stimulates maternal thyroid gland
- v. increase Relaxin secretion from corpus luteum.
- vi. Uterine vasodilatation and myometrial relaxation
- vii. Expansion of decidual NK cells

Increase HCG - Compared to gestation age	Decrease HCG - Compared to gestation age
- Multiple pregnancy	- Failing pregnancy / Abortion
- Erythroblastosis fetalis	- Ectopic pregnancy

- Gestational trophoblastic disease	
- Downs syndrome	

Human placental lactogen -HPL.

00:13:23

- Also called “chorionic growth hormone” because of its similar structure to growth hormone.
- Single Non-glycosylated. “polypeptide” chain
- Molecular weight $\rightarrow 22,279$ Da
- Structurally similar to $\rightarrow$ Growth hormone (96%) and prolactin (67%)
- Before 6 weeks’ source: Both Syncytiotrophoblast and Cytotrophoblast
- After 6 weeks’ source: only Syncytiotrophoblast
- Encoded by: Chromosome 17
- Detectable in maternal serum = 3 weeks after conception
- Peaks in maternal serum = 34-36 weeks of pregnancy
- $T_{1/2} = 10-30$ min
- Production rate = 1g/day
- Concentration: Maternal plasma $>$ Amniotic fluid $>$ Fetal blood & maternal urine

Functions of HPL

- i. Promotes lipolysis (increase circulating serum free fatty acids)
- ii. Decrease leptin secretion
- iii. Increase Insulin secretion – responsible for hyperinsulinemia in pregnancy
- iv. Increases serotonin synthesis (beta cell proliferation → beta cell hyperplasia)
- v. Angiogenic – fetal vessel formation

Functions of other placental hormones

- GnRH: Increase HCG production
- CRH: increase ACTH → cortisol → Initiation of labor,
- HGH: Increase Insulin resistance – HyperInsulinemia. (Human growth hormone variant)
- Neuropeptide Y = increase CRH release.
- Parathyroid releasing protein – Transfer of Ca^{+2} /bone mineralization
- Inhibin → opposes activin
- Activin → Stimulates placental hormone production during pregnancy
- InhibinA – Increased in Down's syndrome
 - Inhibin, Activin – Increased in pregnancies at risk of preeclampsia
 - Inhibin & activin are hormones – Normally secreted by ovaries, corpus luteum, Placenta
- Inhibin – inhibits pituitary – decreased FSH

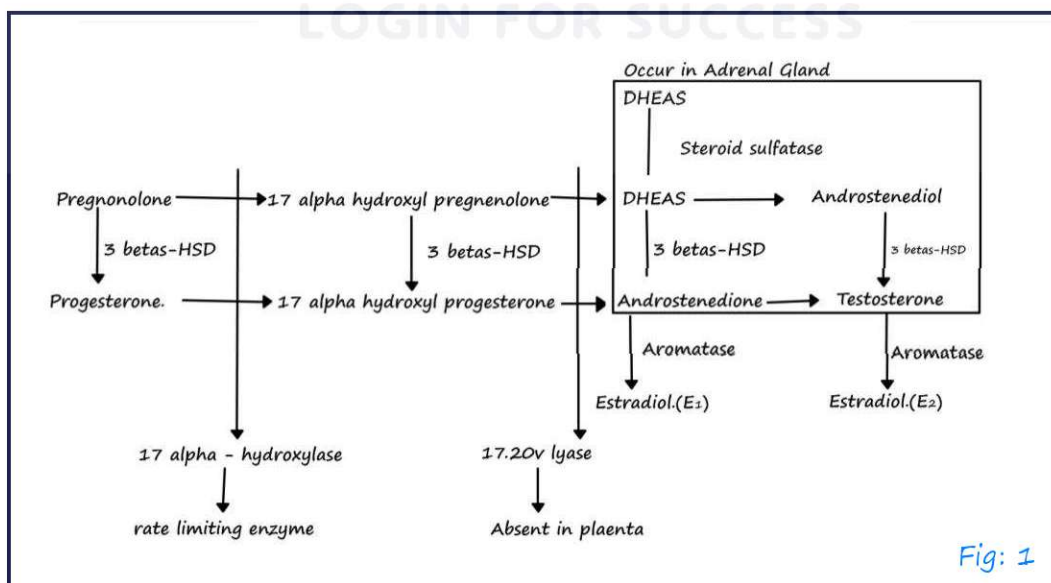
- Activin – activates pituitary – increased FSH

Placental steroids.

00:23:22

- Placental steroids – estrogen & progesterone mainly
- Source 1st trimester – corpus luteum
- Source 2nd trimester – placenta
 - Uteroplacental transition means steroidogenesis from corpus luteum decreases and steroidogenesis from placenta increases
 - Uteroplacental transition begins by – 7 to 10 weeks (8 weeks)
 - Progesterone secretion in placenta begins by = 8 weeks
 - Daily production rate = 250mg (singleton pregnancy)

Pathway of Steroidogenesis



- 3 betas = 3 - beta hydroxysteroid dehydrogenase

- 17,20 lyase = present in fetal adrenal gland
 - Progesterone is directly synthesized in placenta using LDL
 - Estrogen is synthesized in placenta – only by using fetal adrenal androgens because 17,20 lyase is absent in placenta
 - Main precursor of estrogen synthesis in the placenta is $\rightarrow$ DHEA-S
 - 90% of Estrogens
 - 80% of progesterone
 - $E_1 - E_2$ converted to Estriol – E_3 – most important type of estrogen during pregnancy
 - Estetrol $\rightarrow$ metabolite
 - potency $E_2 = 100$ times $> E_1$ 10 times $> E_3$
- Enter maternal circulation

Conditions decreasing placental estrogen production.

- i. IUD – intrauterine death
- ii. Ligation of cord
- iii. Anencephaly
- iv. Fetal adrenocortical insufficiency
- v. Deficiency of steroid sulfatase, 3 beta HSD, Aromatase.
- vi. Down's syndrome
- vii. Maternal glucocorticoid therapy
- viii. Addison's disease

ix. *Gestational Trophoblastic disease (no fetus – no adrenal gland – only progesterone – no estrogen)*

Conditions increasing placental Estrogen production

- *Erythroblastosis fetalis*
- *Maternal androgen producing tumors*



DIAGNOSIS OF PREGNANCY

- Definitive way of diagnosis – Lab test & Ultrasound
- Total duration of pregnancy → 280days (Nageles rule)

(or)

10 lunar months

(or)

9 months + 7days

(or)

40weeks

40weeks



- Fertilization age: 280days – 14
266days (or) 38weeks

Symptoms of pregnancy

00:03:20

- Amenorrhea
- Morning sickness – Due to HCG peaks by 10-12weeks settles by 16weeks
- Frequency of micturition → Increases by 8weeks
- Breast discomfort
- Quickening → 1st fetal movement → Primigravida- 18-20 weeks

Multi gravida -16-18weeks

- Lightening → Sense of relief respiratory distress decrease → 38weeks

Signs of pregnancy

00:08:32

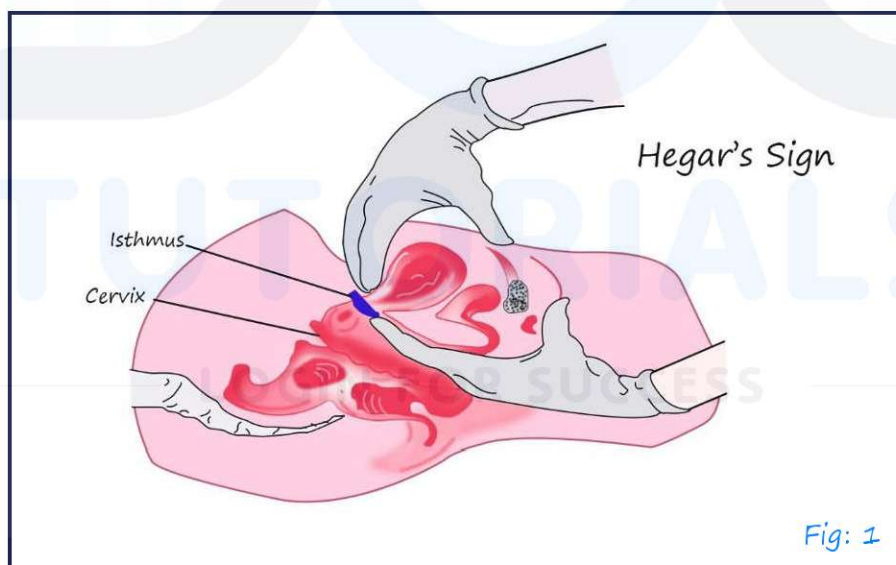
1. Palmer's sign – Rhythmic uterus contraction appears

– as early as 4weeks

- Also seen in – Hematometra

- Pyometra
- IUCD
- Foreign body

2. Hegar's sign: Softening of isthmus



- Appears by 6weeks
- Bimanual flexion of uterus on cervix

3. Goodell's sign

- Appears by 6wks
- Softening of cervix

- Also seen in women using COC (Combined oral contraceptives)

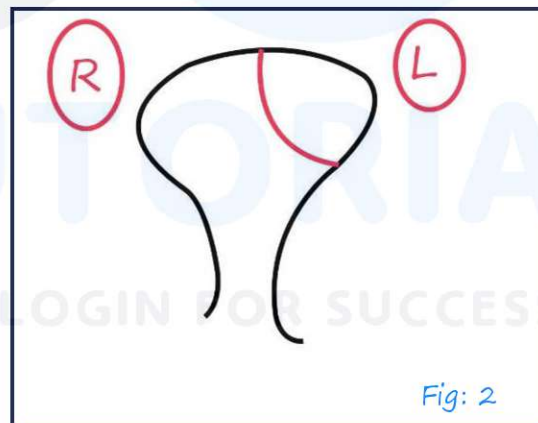
4. Oslander's sign

- Appears by 8wks
- Pulsations felt in lateral fornix
- (Because uterine artery dilated during pregnancy)
- Also seen → PID

5. Chadwick/Jacquemier sign

- Appears by 8wks
- Bluish discoloration of vagina/cervix
- Due to venous congestion
- Also seen in → Fibroid uterus

6. Piskacek's sign



- Appears by 8 weeks
- Lateralisation of fundus
- Also seen in → Fibroid uterus

Breast changes

00:16:35

1. Enlargement with vascular engorgement begins by →
6-8wks

- 2.Secondary areola → 20weeks
- 3.Prominent Montgomery tubercle
- 4.Appearance of colostrum – 12weeks
- 5.Thickening of colostrum – 16weeks

Skin changes

1.Linea nigra

- Linear hyperpigmentation in the midline of abdominal wall extending from pubic symphysis to xiphisternum
- Due to MSH (Melanocyte stimulating hormone)
- appears by → 20weeks

2.Striae gravidarum

- Stretch marks
- White shiny lines on abdominal wall
- Due to breakdown of collagen

3.Chloasma:

- Hyperpigmentation of malar eminence
- Appears by – 24weeks

Fundal height

00:21:21

- Helps in dating pregnancy
- Uterus → Normally pelvic organ
- Crossing pubic symphysis at → 12weeks
- V → Xiphisternum (36weeks)
 - 32weeks and 38 weeks (lightening)

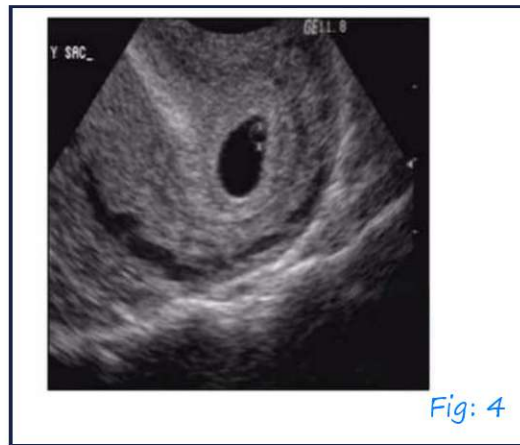
- 28 weeks
- O → Umbilicus (24 weeks)
 - 20 weeks
 - 16 weeks
 - Pubic symphysis (12 weeks)
- External ballottement: Palpating the fetal parts through abdominal wall appears by – 20 weeks
- Internal ballottement – Appears by 16-28 weeks
- Fetal heart sounds
 - Doppler – 10 weeks
 - Stethoscope (or) fetoscope → 20 weeks
- Uterine soufflé – systolic murmur due to increased blood flow in uterine artery
- Funding/foetal soufflé – soft blowing murmurs due to increased blood flow to umbilical artery

Ultrasound

00:33:12

- Most conclusive evidence of pregnancy
- Documenting fetal heart sound by USG





- Yolk sac → Definite sign of intrauterine pregnancy
- Embryo → To check cardiac activity

USG features	Transvaginal scan	Trans abdominal scan
Gestational sac	4wks 5days	5weeks 5days
Yolk sac	5wks 5days	6wsk 5days
Cardiac activity	6wks	7wks

Trimester	Ideal USG parameter for dating pregnancy
1 st trimester	Crown – RUMP length CRL
2 nd trimester	Head circumference (HC) & Trans cerebellar diameter
3 rd trimester	Femur length (FL)

$\text{Crown – RUMP length in Cm} + 6.5 = \text{GA in wks}$

ANTENATAL CARE

Number of antenatal visits

00:01:13

WHO FANC MODEL	2016 WHO FANC MODEL
First trimester	
Visit 1: 8 to 12 weeks	Contact 1 Up to 12 weeks
Second trimester	
Visit 2: 24 to 26 weeks	Contact 2: 20 weeks Contact 3: 26 weeks
Third trimester	
Visit 3: 32 weeks Visit 4: 36-38 weeks	Contact 4: 30 weeks Contact 5: 34 weeks Contact 6: 36 weeks Contact 7: 38 weeks Contact 8: 40 weeks
Return for delivery at 41 weeks if not given birth	

- Minimum number of antenatal visits according to government of India: 4
- According to 2016 WHO FANC model → 8 visits (minimum)

Prophylactic iron therapy

00:04:27

WHO Recommendation	MOHFW Recommendation
Start iron from 1 st visit	Start at 14 weeks
Elemental Iron – 60 mg ferrous salt (For at least 6 months during pregnancy and 3 months postpartum)	Elemental Iron – 100 mg (335 mg of ferrous salt)
400 micrograms of folic acid	500 micrograms of folic acid
Albendazole 400 mg or Mebendazole 500 mg (Single dose in the second trimester)	

Folic acid

00:08:03

- Should be ideally started 3 months prior to planned pregnancy
- Prophylactic dose 0.4 mg = 400mcg
- Therapeutic dose – 4 mg
- Indications of therapeutic dose
 1. Megaloblastic anemia
 2. Hemolytic anemia
 3. Overt diabetes
 4. Anti-epileptic drugs
 5. Previous H/O neural tube defects

Q. A pregnant woman at 12 weeks gestation is bitten by rabid dog. What is the ideal next step?

- A. Only vaccine
- B. Only immunoglobulin
- C. Wound care
- D. Vaccine + Immunoglobulin ✓

Vaccines in pregnancy

00:11:12

- Live Attenuated Virus Vaccines

Immunobiological Agent	Indications for immunization during pregnancy	
Measles	contraindicated	Vaccinate postpartum Breastfeeding not a contraindication
Mumps	contraindicated	Vaccinate postpartum
Rubella	contraindicated	Vaccinate postpartum
Poliomyelitis oral = live attenuated;	Not routinely recommended	Vaccine women traveling in endemic areas

injection = enhanced potency inactivated virus		
Yellow fever	Travel to high-risk areas	Limited theoretical risk outweighed by risk of yellow fever
Varicella	contraindicated	Vaccination postpartum
Smallpox (Vaccinia)	contraindicated	Only vaccine known to cause fetal harm

- All live attenuated vaccines are avoided in pregnancy
- After taking any live vaccine, pregnancy should be postponed for at least 1 month (If possible – 3 months)
- Inadvertent exposure during pregnancy → No need to terminate the pregnancy

Other

<i>Immunobiological Agent</i>	<i>Indications for immunization during pregnancy</i>	
Influenza	All pregnant women, regardless of trimester during flu season	Inactivated virus vaccine
Rabies	Indications for prophylaxis not altered by pregnancy	Killed virus vaccine
Human Papillomavirus	Not recommended	No teratogenicity
Hepatitis B	Preexposure and postexposure for women at risk of infection	Used with hepatitis B for some exposures
Hepatitis A	Preexposure and postexposure if at risk	Inactivated virus

Inactivated Bacterial Vaccines

<i>Immunobiological Agent</i>	<i>Indications for immunization during pregnancy</i>
<i>Pneumococcus</i>	<i>Indications not altered by pregnancy</i>
<i>Meningococcus</i>	<i>Indications not altered by pregnancy</i>
<i>Typhoid</i>	<i>Not recommended routinely</i>

Toxoids

<i>Immunobiological Agent</i>	<i>Indications for immunization during pregnancy</i>
<i>Toxoids (Tetanus diphtheria acellular pertussis)</i>	<i>Recommended in every pregnancy, preferably between 27 & 36 weeks</i>

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Immunoglobulins

<i>Immunobiological Agent</i>	<i>Indications for immunization during pregnancy</i>	
Hepatitis B	Postexposure prophylaxis	With hepatitis B virus vaccine
Rabies	Postexposure prophylaxis	With rabies killed virus vaccine
Tetanus	Postexposure prophylaxis	With tetanus toxoid
Varicella	Postexposure prophylaxis	Indicated also for newborns or women who developed varicella within 4 days before delivery or 2 days following delivery
Hepatitis A virus vaccine should be used with hepatitis A immunoglobulin	Postexposure prophylaxis	Within 2 weeks of exposure

Recommended daily dietary allowances 00:21:15

Nutrient	Pregnancy	Location
Fat soluble		
Vitamin A	770 micg	1300 micg
Vitamin D	15 micg	15 micg
Vitamin E	15 micg	19 micg
Vitamin K	90 micg	90 micg
Water soluble		
Vitamin C	85 mg	120 mg
Thiamine	1.4 mg	1.4 mg
Riboflavin	1.4 mg	1.6 mg
Niacin	18 mg	17 mg
Vitamin B6	1.9 mg	2 mg
Folate	600 micg	500 micg
Vitamin B12	2.6 micg	2.8 micg
Minerals		
Calcium	1000 mg	1000 mg
Sodium	1.5g	15g
Potassium	4.7.g	5.1g
Iodine	220micg	290micg

- Additional calorie requirement
 - 1st trimester – 85 kcal / day
 - 2nd trimester – 285 kcal/day
 - 3rd trimester – 475 kcal/day

Antenatal investigations

- Hb and Hematocrit
- Blood grouping and typing
- Blood glucose
- VDRL and HIV
- HBsAg
- Urine microscopy
- Urine culture

Other common investigations

- Serum TSH
 - Rubella IgG and IgM
 - Screening for hemolytic anemia
- Optional but are usually done

Ultrasound scans

1. Booking visit / confirmatory scan: - done at 6 weeks
2. First trimester screening + NT → done at 11-14 weeks - Q. 12 weeks
3. TIFFA (anomaly scan) → Targeted imaging for foetal anomalies, ideally done at 20 weeks
4. Growth scan
 - If required, fetal echo done at 24 weeks (also uterine artery doppler)
 - ↓
 - Indications

- Family H/O congestive heart disease
- Past H/O congestive heart disease
- Mother has congestive heart disease
- Overt DM
- Abnormality in TIFFA Scan
- Uterine artery doppler is to predict IUGR at 24weeks
- Growth scan at 32 weeks and 38 weeks

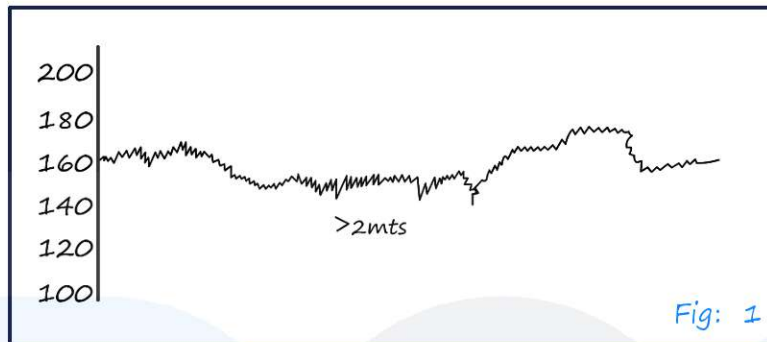


NST AND CTG

NST (Non stress test)

00:00:30

- Recording FHR without uterine contractions



CTD

Cardiotocography

- Recording FHR with uterine contractions

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Living ligatures

Blood vessels running with in the layers of myometrium

Occlusion of vessels occurs during myometrial contractions

Interpretation of CTG

00:02:37

- Baseline heart rate
- Baseline variability
- Acceleration
- Deceleration

Normal Baseline Heart Rate

00:02:53

- Rounded to increments of 5 bpm during a 10mt segment for a minimum of 2 minutes
- Normal – 110-160bpm
- Tachycardia – > 160bpm
- Bradycardia – < 110bpm

Causes of bradycardia	Causes of tachycardia
Congenital heart block	Maternal fever
Fetal distress	Cardiac arrhythmias
Abruption	Sympathomimetic
Maternal hypothermia	Atropine

Baseline Variability

00:06:37

- Fluctuation in baseline heart rate with irregular amplitude & frequency
- Minimal ≤ 5 bpm
- Moderate (normal) – 6-25bpm
- Marked > 25bpm

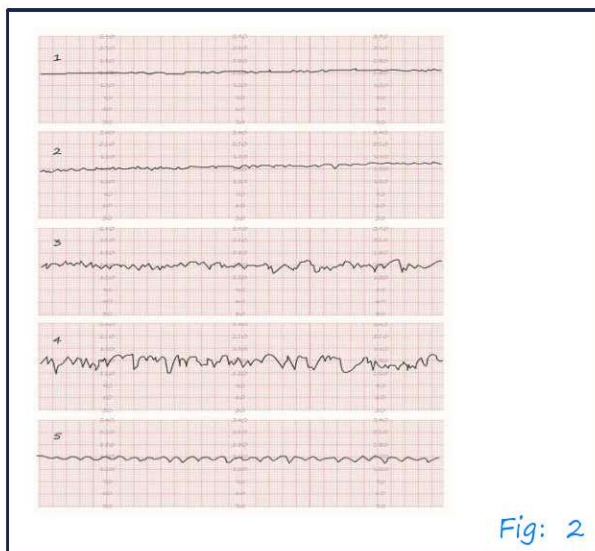


Fig: 2

- Increased variability is associated with fetal activity
- **Decreased variability is associated with**
 - Barbiturates
 - Anesthetic drugs
 - Corticosteroids
 - Meperidine
 - Buprenorphine
 - Mgso₄
 - Fetal acidemia
 - Maternal acidemia
 - DKA
 - **Reduced baseline HR variability is the single most reliable sign of fetal compromise**

Acceleration

00:13:13

- Abrupt increase in HR
- Onset to peak < 30sec
- Increases by at least 15 bpm
- Duration at least 15sec to 2min
- Lasting for > 2min to < 10min → prolonged acceleration
- Lasting for > 10min → baseline change 10bpm/10sec for < 32weeks

Reactive NST

00:20:06

- Baseline HR 110 to 160 bpm
- Baseline variability 5 to 25 bpm
- 2 or more accelerations in 20 minutes each persisting for more than 15 seconds and the increasing by more than 15bpm
- No deceleration

Deceleration

00:20:21

- Fall in heart rate - 3 types
- Early declaration
- Late declaration
- Variable declaration
 - i. Uncomplicated
 - ii. Complicated/typical

Early Deceleration

00:29:08

- Uniform/symmetrical
- Repetitive
- Starts early in the contractions
- Returns to baseline at the end of contractions
- They do not drop to more than 40 beats from the baseline heart rate
- Due to head compression
- Both early and late deceleration are not abrupt, repetitive or uniform, related to contractions

Late Decelerations

00:30:36

- It comes after contractions
- It is uniform /symmetrical
- Repetitive
- Onset at the mid to end of contractions
- Nadir more than 20 seconds after the peak of contraction
- Late deceleration is due to fetal hypoxia (preeclampsia, DM/1UGR, Placental insufficiency)

Variable Decelerations

- Variable declaration is sudden/rapid/abrupt / not repetitive or related to contractions
- Uncomplicated (due to cord compression/occlusion)
- Complicated/atypical (complete cord delusion)

Uncomplicated Variable Declaration

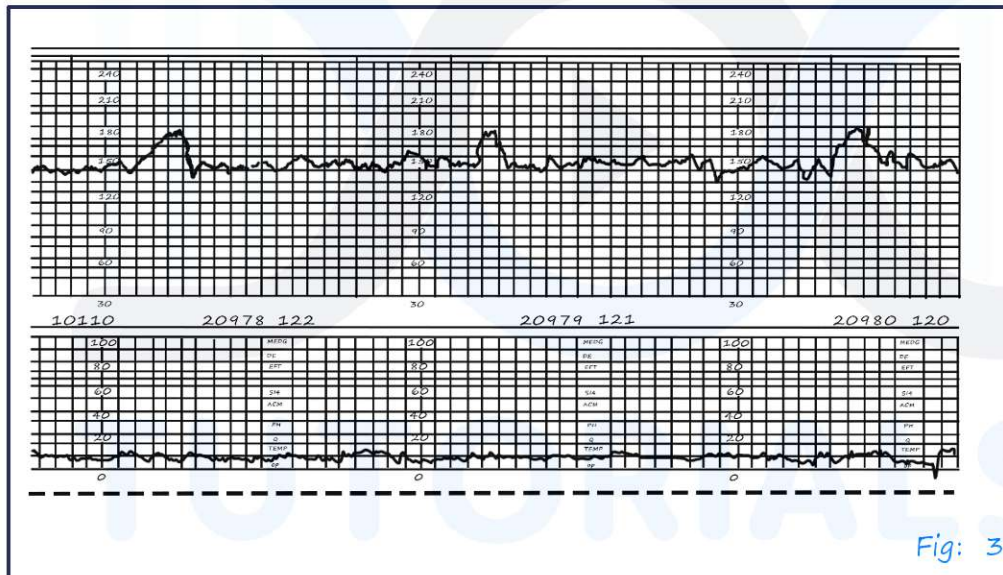
00:34:56

- Variable (vary with successive contraction)
 - Intermittent
 - Sudden rapid fall
 - Quick recovery
- } Abrupt (onset to nadir <30s)
- Shouldering – It is increase in heart rate either before or after deceleration and it occurs due to partial deceleration

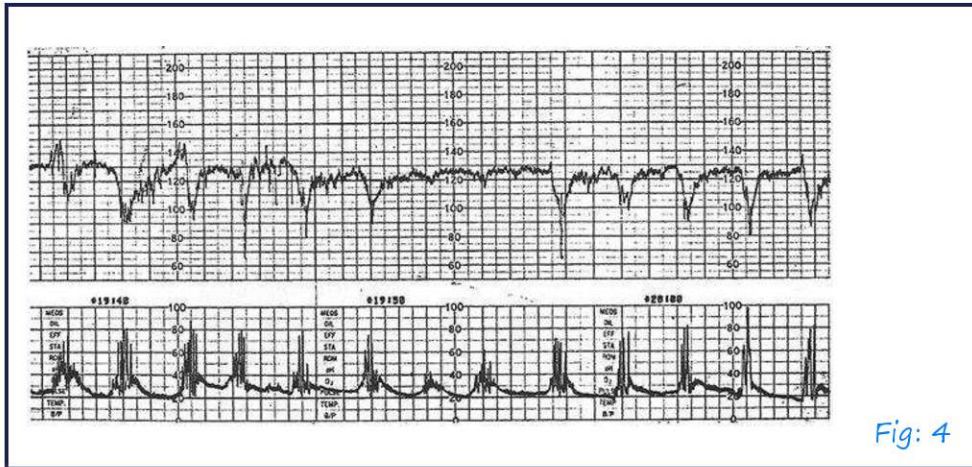
Complicated Atypical Variable Declaration

00:37:13

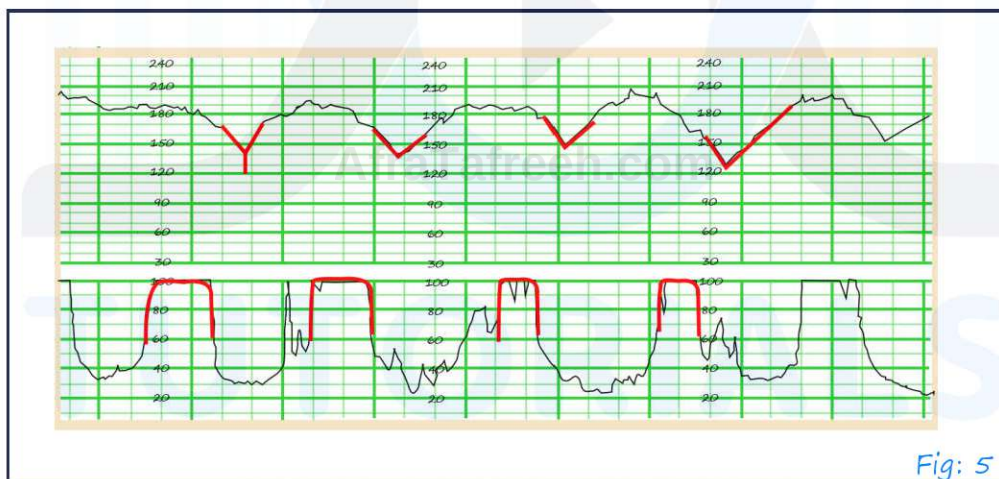
- Baseline tachycardia /baseline bradycardia
- Absent variability
- Not related to contraction
- No shouldering
- Complete occlusion
- Fall in heart rate by > 60 bpm/absolute heart rate touching 60bpm



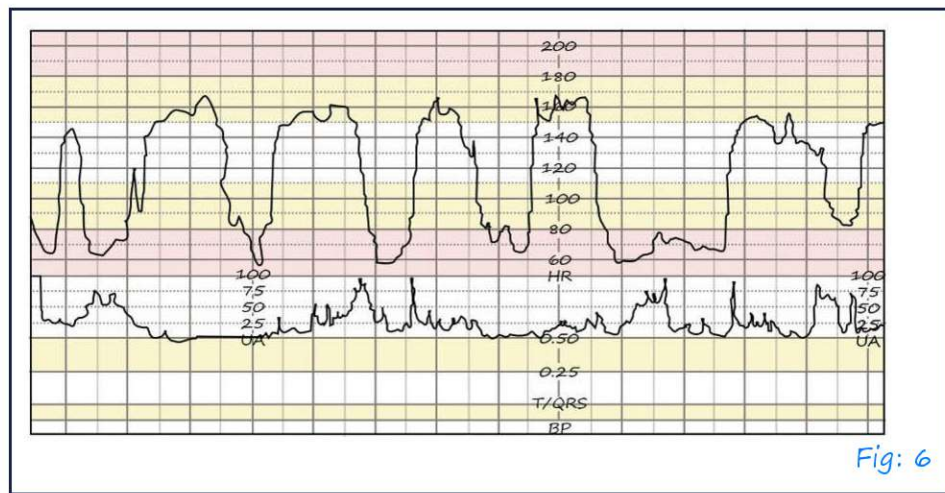
- Interpret the above picture
- Baseline > 150
- Variability - 10bpm
- Acceleration - present
- Deceleration - absent
- Reactive NST



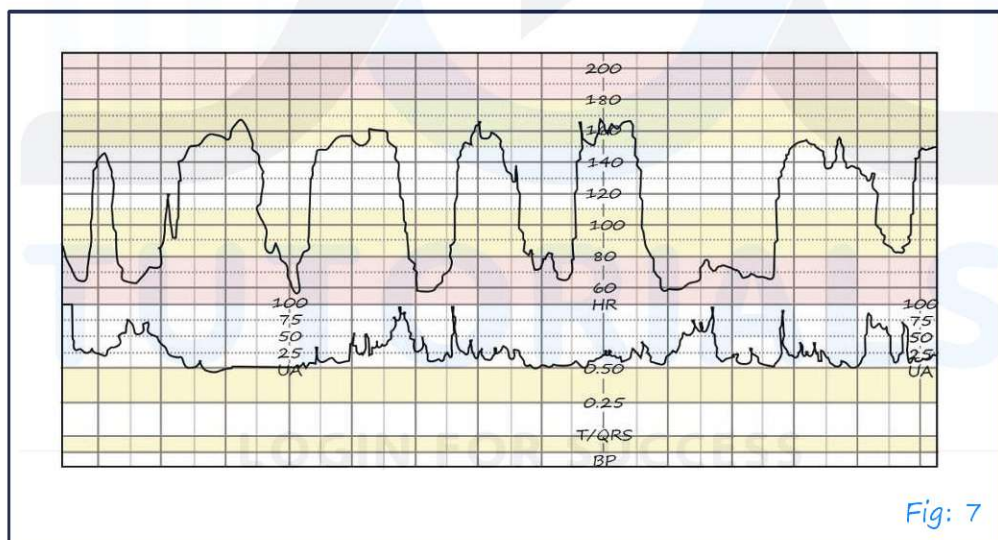
- The above deceleration is due to
- Early deceleration
 - i. Head compression



- Interpret the above picture
 - Late decelera
- Interrupt the given CTG



- Variable deceleration
- Shouldering is present-increase in heart rate after deceleration is shouldering
- It is an uncomplicated variable deceleration.
- Interrupt the below picture

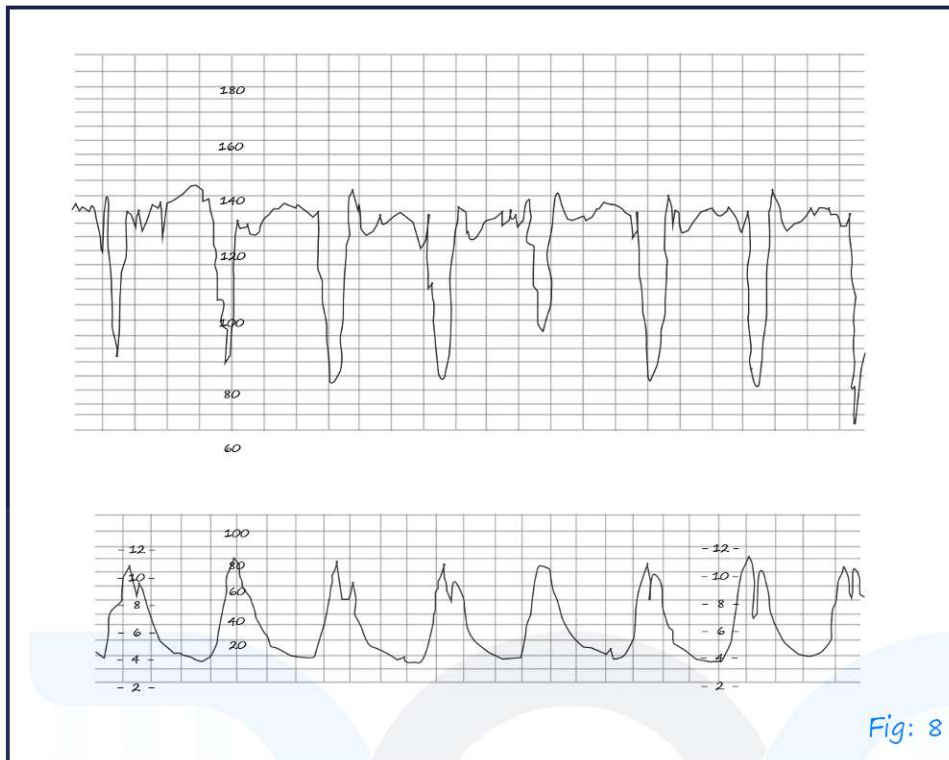


- Atypical variable declaration

Prolonged Declaration

00:49:29

- Drop in the FHR less than 80bpm for more than 3minutes
- The below CTG is due to



- Sinusoidal Pattern – due to fetal anemia/ hydrops fetalis

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Sinusoidal Pattern

- Regular oscillations fixed of the baseline rate
- Markedly reduced baseline variability
- Resembles sine wave
- 5 to 15bpm
- 3 to 5 cycles per minute
- Persisting for at least 20minutes
- Sinusoidal pattern is due to fetal anemia

NST

3 CATEGORIES

- Category 1- Normal

- Category II- Indeterminate
- Category III- Abnormal

• Category 1 – Normal • Baseline rate – 110-160bpm • Baseline FHR variability – moderate • Late or variable deceleration absent • Early deceleration present or absent • Accelerations – present or absent	• Category III – abnormal • Include either Baseline FHR variability and any of the following • Recurrent late decelerations • Recurrent variable decelerations • Bradycardia • Sinusoidal pattern
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TUTORIALS

LOGIN FOR SUCCESS

BIOPHYSICAL PROFILE

Components

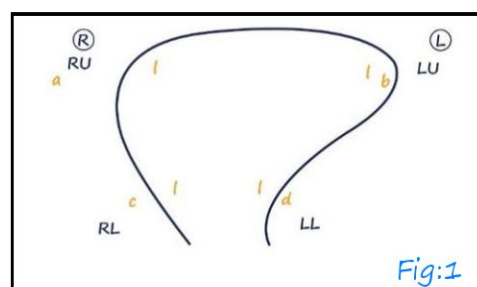
00:00:58

- Non stress test
- Fetal breathing
- Fetal body movements
- Fetal tone
- Amniotic fluid

Score of BPP

00:02:24

1. NST → reactive means ≥ 2 acceleration of ≥ 15 bpm for at least 15 seconds within a 20-40 minute
2. Fetal breathing (USG) – ≥ 1 episode of breathing movement lasting for ≥ 30 seconds
3. Fetal body movement: – ≥ 3 discrete body movements
 ≥ 1 episodes of extremity extension and return to flexion
4. Fetal tone: – Flexion and extension movements ≥ 1 episodes of Extremity extension and return to flexion
5. Amniotic fluid volume: – Single deepest pocket ≥ 2 cm



- $a+b+c+d = AFI$
- Single deepest pocket - biggest of 4

	Reactive	Non-reactive
Non stress test	2	0
Fetal breathing	2	0
Fetal body movements	2	0
Fetal tone	2	0
Amniotic fluids volume	2	0

Maximum score - 10

Minimum score - 0

Possible scores: - 10, 8, 6, 4, 2, 0

Biophysical profile score	Interpretation	Recommended Management
10	Normal, Nonasphyxiated fetus	No fetal indication for intervention; repeat test weekly except in diabetic patients and post term pregnancy (twice weekly)

8/10 (Normal AFV) 8/8 (NST not done)	Normal, Nonasphyxiated fetus	No fetal indication for intervention; repeat testing per protocol
8/10 (Decreased AFV)	Chronic fetal asphyxia suspected	Deliver
6	Possible fetal asphyxia	If amniotic fluid volume abnormal, deliver If normal fluid at > weeks with favourable cervix, deliver If repeat test ≤ 6 deliver If repeat test > 6 observe and repeat per protocol
4	Probable fetal asphyxia	Repeat testing same day; if biophysical profile score ≤ 6 , deliver
0 to 2	Almost certain fetal asphyxia	Deliver

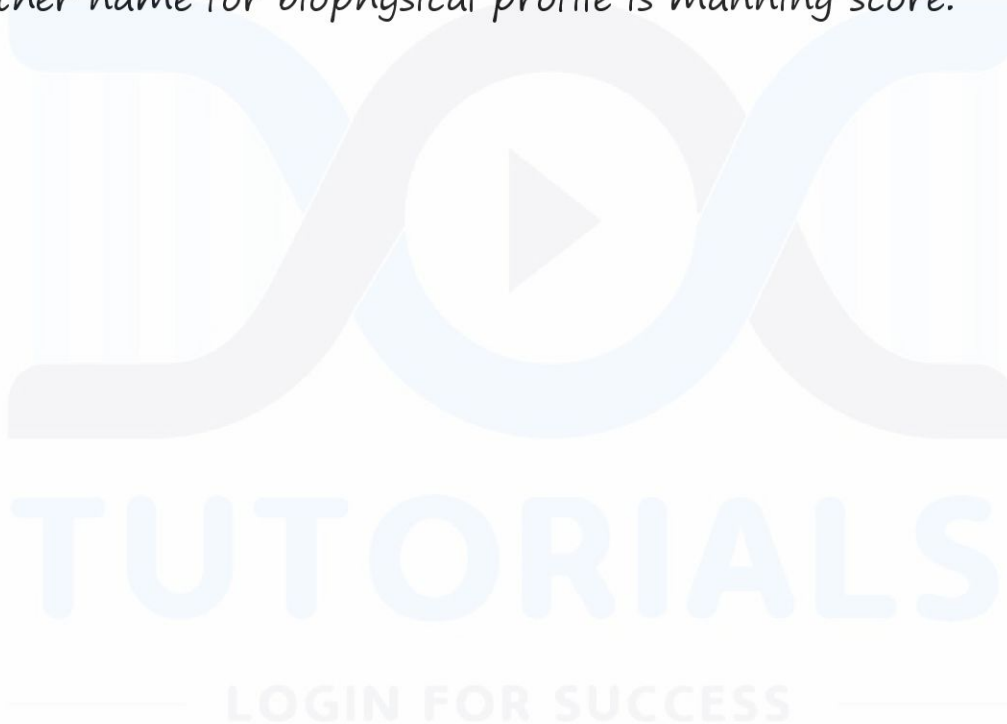
- Fetal tone begins by 7-8
- Fetal movements by 9 weeks
- Breathing movements by 20-21 weeks
- Heart rate reactivity by 26-28 weeks

- In the presence of progressive hypoxia reactivity in NST and fetal breath movements are the first biophysical profile to disappear followed by cessation of fetal movements and finally fetal tone.

Modified BPP

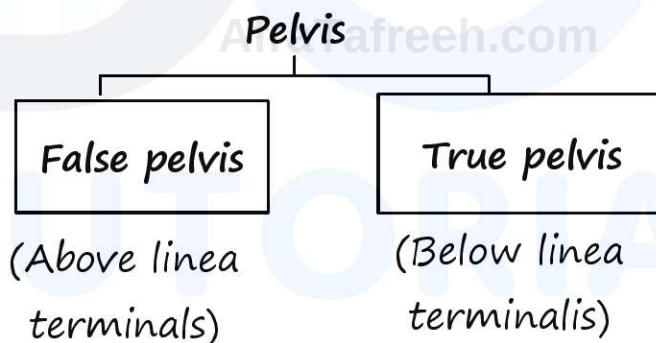
00:16:15

- NST
- AFI
- Other name for biophysical profile is manning score.



MATERNAL PELVIC AND FETAL SKULL

- Consist of 4 bones – 2 innominate bones (ilium, ischium, Pubis)
 - 1 Sacrum
 - 1 coccyx
- 4 joints → pubic symphysis between 2 pubic bones
 - Sacroiliac joint (synovial joint)
 - Sacro coccygeal joints (synovial joint)
 - Symphysis pubis (fibro cartilaginous joint)



- Both are separated by imaginary circle called “Pelvic Brim” and it is formed by
 - i. Pubic symphysis
 - ii. Pubic crest
 - iii. Pubic tubercle
 - iv. Pectineal line
 - v. Ilio pubic eminence

vi. Ilio pectineal line

vii. Sacroiliac joint

viii. Sacral Ala

ix. Sacral promontory

- All together form linea terminalis
- False pelvis is formed by 2 iliac bones on either side
- Anteriorly, it is devoid of bone. It accommodates growing uterus and cushions the uterus
- It does not take part in labor

True pelvis

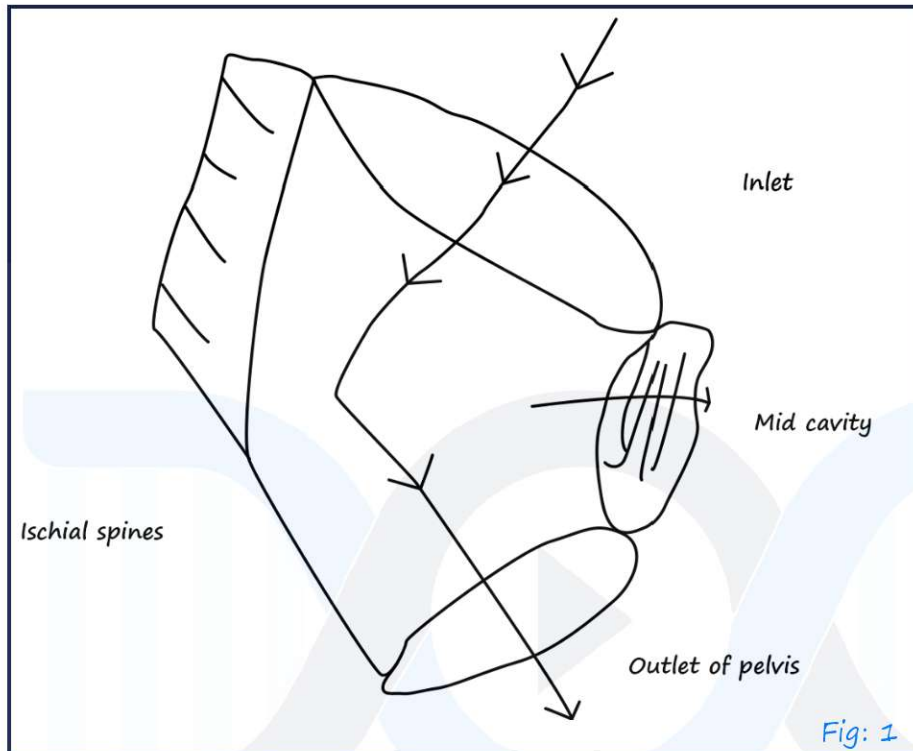
i. Inlet of pelvis □ formed by linea terminalis/ pelvic Brim

ii. Mid pelvis

iii. Outlet of pelvis

- Lower most part of pelvis with Ischial tuberosities on either side
- Lower border of pubic symphysis anteriorly, coccyx posteriorly
- The area between Inlet and outlet of pelvis is mid pelvis
- Pelvic cavity is not straight line, instead it converges at one point and then diverges downwards and outwards

- That point is "Ischial spines" which is landmark for mid pelvis



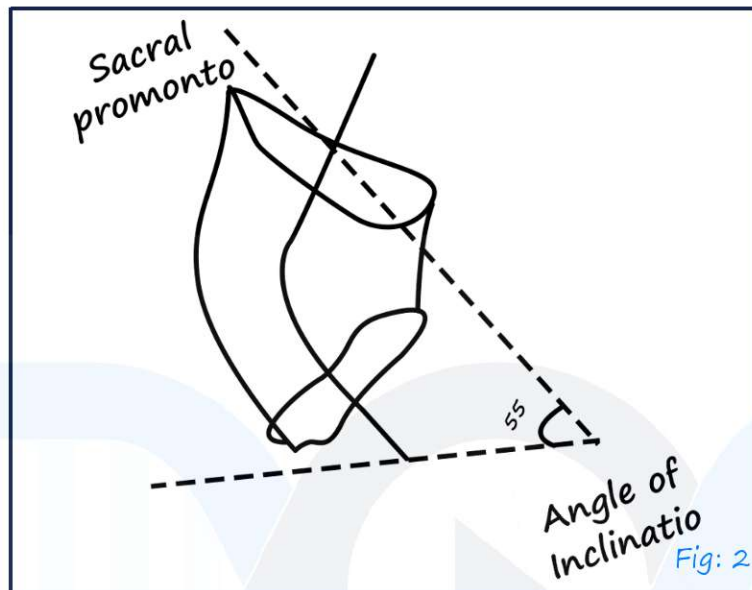
Importance of ischial spines

- Narrowest diameter of mid pelvis
- Fetal head is said to be engaged once it crosses ischial spines
- Pudendal nerve is present, so pudendal block given at that point
- Pelvimetry (measurement of pelvis is done at that point)

Angle of Inclination & sacral angle

- Angle made by the pelvic cavity with horizontal is 55° called angle of Inclination

- ii. Sacral angle is the angle between curve of inlet and first sacral vertebrae measurement $\approx 90^\circ$



High inclination

- Due to sacralization of fifth lumbar vertebra - L5 fused with sacrum

Obstetric Significance

- Angle of inclination increases
- Delay in engagement of head
- Difficulty in descent and Internal rotation

Depth of pelvis

- Length of Anterior segment $\rightarrow$ 4 cm (pubic symphysis)

- Length of posterior segment $\rightarrow$ 11.5 cm (sacral promontory to tip of coccyx)

Diameters of Pelvis

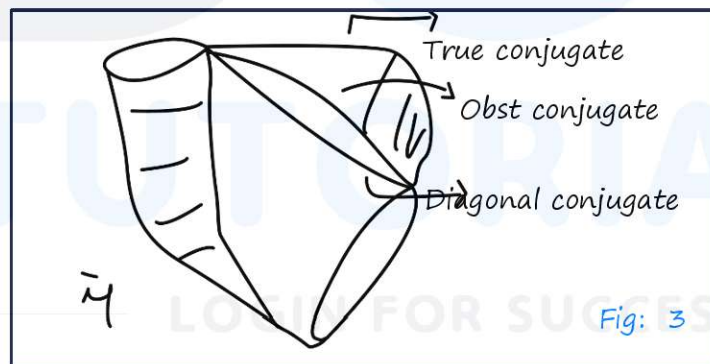
00:12:50

a. Anteroposterior (AP) diameter

- Inlet $\square$ Distance between pubic symphysis and sacral promontory

Anatomical conjugate

- True conjugate (Anatomical conjugate) $\rightarrow$ 11cm (distance between upper part of pubic symphysis and sacral promontory)



- Obstetric conjugate $\rightarrow$ 10 cm (distance between upper most part of pubic tubercle and sacral promontory)

It is an angle of significance in obstetrics.

- iii. Diagonal conjugate = 12cm (distance between lower part of pubic symphysis and sacral promontory makes diagonal conjugate)
- It is the only clinically measurable angle by pelvic examination.

OBS conjugate = Diagonal conjugate - 1.5 to 2cm

b. Transverse diameters of Inlet

- Obtained by joining the two farthest points on lateral wall of pelvis

T.D - 13.5cm

c. Oblique diameter

- Obtained by joining sacroiliac joints on one side with iliopectineal eminence on other side - 12cm
- Right oblique diameter is named after right sacroiliac joint

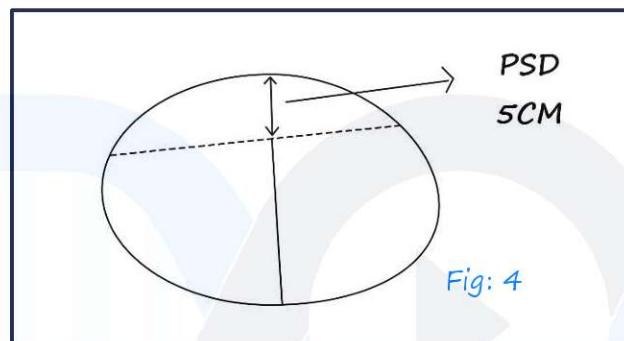
d. Sacro cotyloid diameter

- Obtained by joining sacral promontory and iliopectineal eminence - 9.5cm
- Rt diameters corresponds to right iliopectineal eminence
 - Posterior sagittal diameter

- Obtained by joining sacral promontory with point of intersection of transverse and AP diameters of Inlet

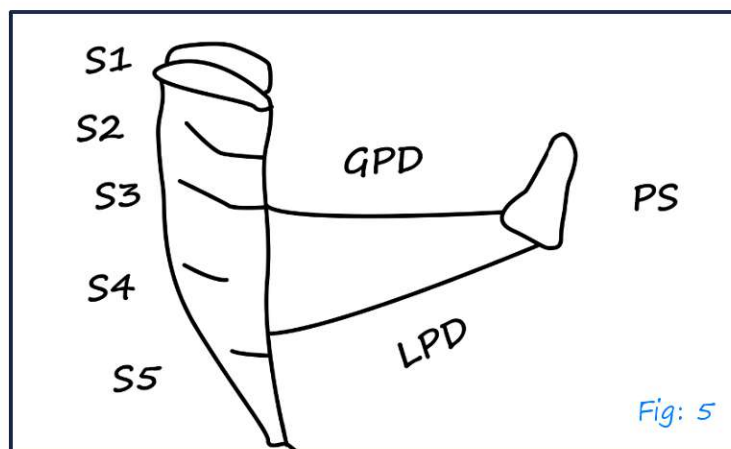
$$PSD = 5\text{cm}$$

- It accommodates occipital bones of fetus



Mid pelvis diameters

- Area between inlet and outlet
- 1. Plane of Greater pelvis dimension
 - Obtained by joining the pubic symphysis (mid-point) and junction between S_2, S_3
 - It is biggest plane in entire pelvis



11. Least pelvic dimension (LPD)

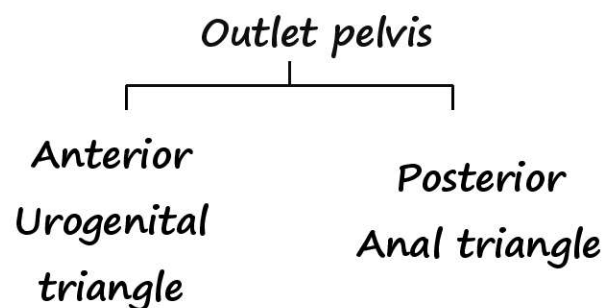
- Obtained by joining lower part of pubic symphysis with S_4, S_5 Vertebra
- It makes AP diameter of mid pelvis
- Transverse diameter at that level crosses the ischial spines which is called transverse dm of mid pelvis or inter spinous diameter (ISD)

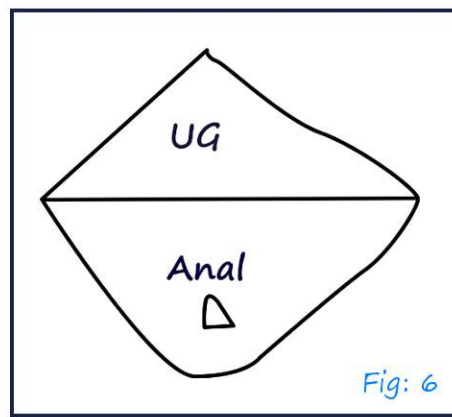
ISD 10.5 cm

- It is shortest transverse diameter of entire pelvis

Pelvic outlet

- Anteriorly → Lower part of pubic symphysis
- Laterally → ischial tuberosities
- Posteriorly → tip of coccyx
- Line joining ischial tuberosities divides outlet into





Diameters

i. Transverse diameter of outlet



Inter tuberos diameter = 11cm

ii. AP diameters of outlet = 11 to 13cm (variable due to coccyx)

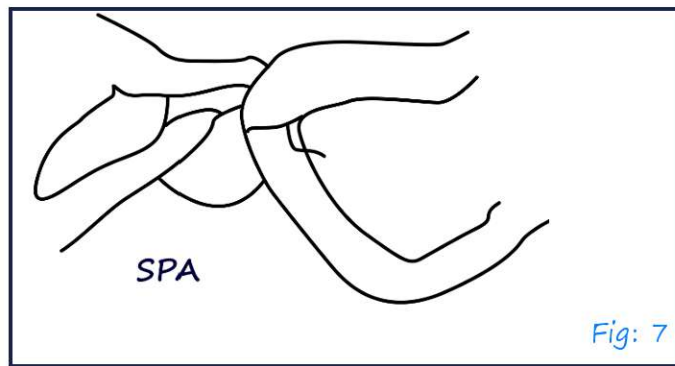
- Standing position □ 11cm
- Lithotomy position □ 13cm

iii. Posterior sagittal diameter = 8.5cm (distance between tip of coccyx & point of Intersection of AP & transverse diameters)

LOGIN FOR SUCCESS

Sub pubic angle

- Angle made by inferior pubic rami on either side with pubic symphysis
- SPA = 85° for gynecoid pelvis
- Platypelloid pelvis - SPA $> 90^{\circ}$ (wide obtuse angle)



Waste space Morris

- Distance between pubic symphysis and fetal head during labor
- It should not exceed 1cm, otherwise pelvis is inadequate

Normally < 1cm

III types of pelvis

00:37:13

Caldwell moly classification

- Gynecoid (50%) – most common
- Anthropoid (25%)
- Android (20%) – male type pelvis
- Platypelloid (5%) – least common (flat pelvis)

	<i>Gynecoid</i>	<i>Android</i>	<i>Anthropoid</i>	<i>Platypelloid</i>
<i>Inlet</i>	<i>Oval / Round</i>	<i>Heart shape</i>	<i>Vertically oval</i>	<i>Transversely oval</i>
<i>Sidewalls</i>	<i>Parallel</i>	<i>Convergent</i>	<i>Divergent</i>	<i>Divergent</i>
<i>Ischial spines</i>	<i>Blunt</i>	<i>Prominent</i>	<i>Blunt</i>	<i>Blunt</i>
<i>Subpubic angle</i>	<i>Wide</i>	<i>Narrow</i>	<i>Narrow</i>	<i>Wide</i>
<i>Sacrosciatic notch</i>	<i>Wide</i>	<i>Narrow</i>	<i>Wide</i>	<i>Narrow</i>
<i>Labor</i>	<i>Favours Occipito Anterior</i>	<i>Obstructed labor</i>	<i>Occipito posterior</i>	<i>Occipito posterior</i>

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Fetal Skull

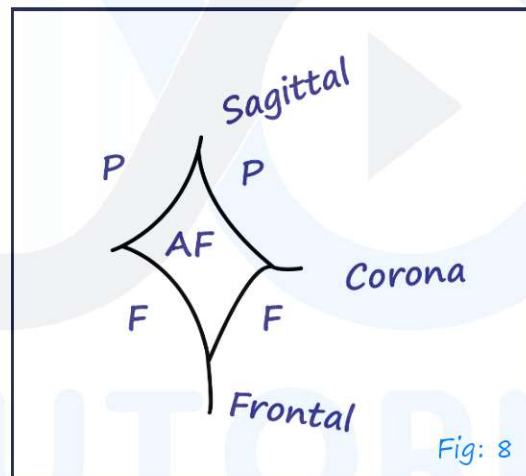
00:43:35

- 4 sutures
 - i. Sagittal suture between two parietal bones on either side
 - ii. Coronal suture between one frontal and one parietal bone
 - iii. Frontal suture between 2 frontal bone (metopic suture)
 - iv. Lambdoid suture between parietal & occipital bones

Fontanelles

a. Anterior fontanelle

- Between two frontal & two parietal bones
- Bregma
- Diamond / Rhomboid shape
- Measure 3x3cm
- Ossifies at 18 months
- Its position in P/V examination helps to identify fetal position



b. Posterior fontanelle

- Triangular shape
- 2 parietal bones and occipital bone
- Known as lambda
- Measure 1.2 x 1.2cm
- Ossify at birth or before 6m
 - Fontanelles helps to identify attitude of baby (either flexion or extension of head during vaginal examination)

- i. If you feel posterior fontanelle → flexion of fetal head
- ii. If you feel anterior fontanelle → partial extension of head / military position leads to Brow presentation
- iii. If you feel chin / facial parts → extension of fetal head

Vertex

- i. Area between anterior & posterior fontanelle
- ii. Quadrangular area
 - Boundaries → Anterior – Bregma and coronal sutures
 - Posterior – lambda
 - Laterally – parietal eminence

Brow

- Boundaries
 - Anterior fontanelle, coronal suture, root of nose & supraorbital ridges.

Face

- Boundaries
 - Areas between supraorbital ridge & submental area of neck

Attitude of Baby

- Complete Flexion – Vertex presentation
- Complete extension – Face presentation
- Partial extension / Incomplete extension – Brow presentation

Diameters of Skull

i. AP Diameters

		Attovolt	Presentati on
Sub occipito Bregmatic (SOB)	9.5cm	Complete flexion	Vertex
Suboccipito frontal (SOF)	10.5	Incomplete flexion	Vertex
Occipito frontal (OF)	11.5	Deflexed head	Vertex
Mento vertical	14	Partial extension (Neutral position)	Brow
Subment to (SMB) Bregmatic	9.5	Complete extension	Face

- Mode of delivery in brow presentation is C-Section

Transverse Diameters

- i. Biparietal Diameter – 9.5cm
- ii. Super sub parietal – 8.5cm
- iii. Bi temporal diameter – 8cm
- iv. Bi Mastoid Diameter – 7.5cm

Engaging Diameter

- The transverse diameter of skull which enters pelvis is always BPD – 9.5cm (Bi parietal diameter)

Diameter of engagement

- The pelvic diameter through which BPD enters the pelvis

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TUTORIALS

LOGIN FOR SUCCESS

PHYSIOLOGY OF LABOR

- Myometrium has smooth muscle cells with little connective tissue
- Smooth muscle cells are arranged in 3 layers
- Outer longitudinal
- Inner-oblique & transverse (2 layers)
- Both hypertrophy & hyperplasia are responsible for enlargement of uterus during pregnancy
- More hypertrophy than hyperplasia
- Volume increases 1000-fold

Myometrial Contractility increases with

- i. Increases in gap junction (with increase in connexin 43 protein)
- ii. Increase in oxytocin receptors

Factor Associated with Onset of Labour

- Estrogen
- Prostaglandins
- Peptides
- Oxytocin
- Relaxin
- VIP/prolactin/CRH
- Interleukin/cytokines (IL₁, IL₆, IL₈, TNF, Alpha)

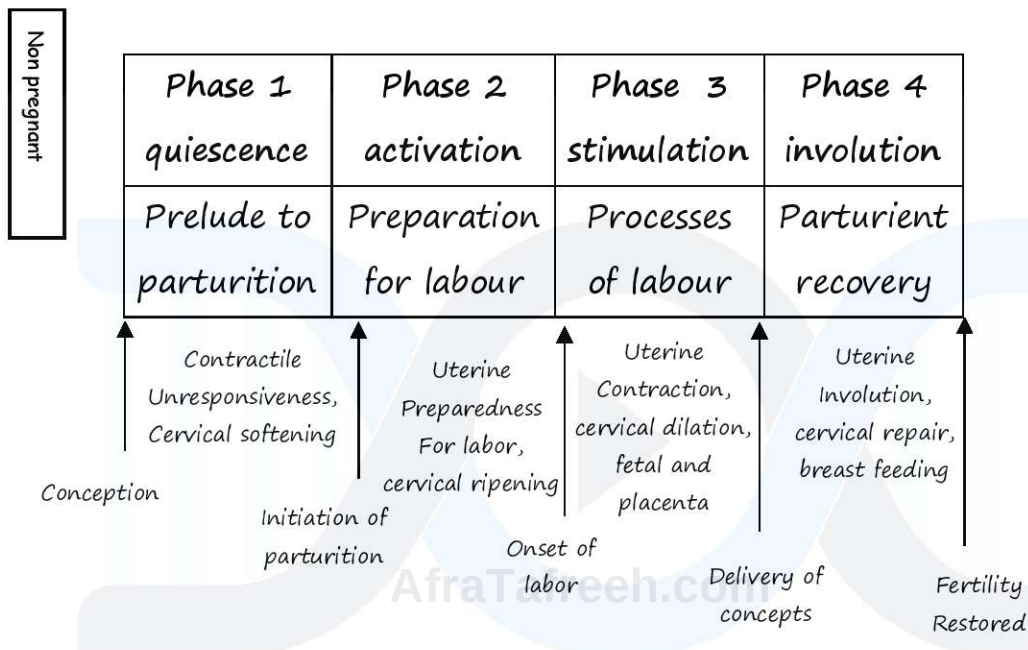
Hormone Opposing Uterine Contraction →

Progesterone

Phases of Labour

00:05:43

- There are four phases
- There are four phases



- Phase 1: Prelude
- Phase 2: Preparation
- Phase 3: Process of labor (stage 1, 2, 3 of Labour)
- Phase 4: Involution (Recovery) → stage 4 of labour

Gap Junction

- Between smooth muscle cells of myometrium, there are proteins known as connexin 43 in uterus

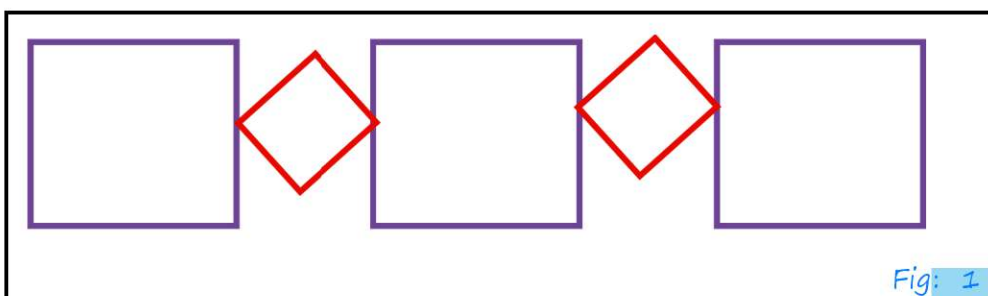
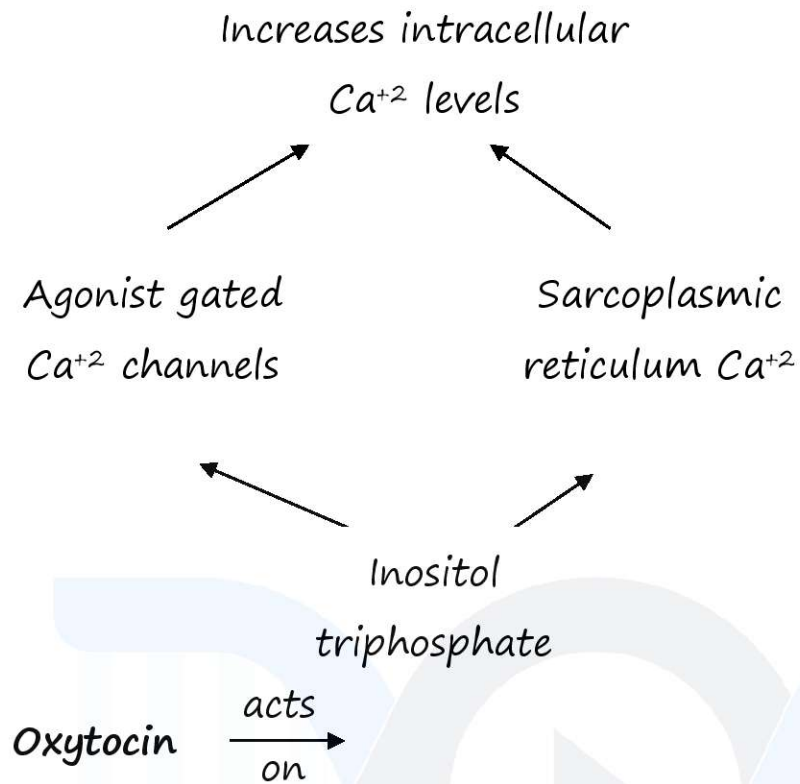


Fig: 1

- Connexion 43 allows rapid transmission of electrical signals
- Development of gap junctions is one of the earliest change in prelabour
- **It occurs in phase 2 of labour- preparatory phase**
- E_2 & PG: increases gap junction formation
- progesterone decreases gap junction formation
- Myometrium is poorly innervated
- No constant pacemaker in uterus and each cell is capable of becoming a pacemaker

Estrogen	Progesterone
- Increases Gap Junction - Induces Oxytocin receptors - Decreases RMP (Resting membrane Potential) - Sensitises the myometrium to alpha adrenoreceptor to favour uterine contraction - Increases PG synthesis	- Decreases Gap junction - Inhibit formation of oxytocin receptors - Increases RMP - Blocks alpha adrenoreceptors - Decreases PG synthesis



	Non-pregnant	Pregnant
Cervix	Firm	Soft
Myometrium	Quiescent	Excitable at term

- Prostaglandins & collagenases/GAG help in ripening of cervix

Final common pathway in initiation of labour

Prostaglandins

- Amnion & decidua



- Produce PGE_2 & $\text{F}_2\alpha$



- Increases sensitivity of myometrium to oxytocin receptors



- Oxytocin increases synthesis of PG



- More formation of gap junction



- More increases Ca^{+2}



- Contractions

- Collagenase

- Cervix has

i. Type I Collagen (66%)

ii. Type III Collagen (33%)

Dermatan & chondroitin (hydrophobic)



Replaced by hyaluronic acid (hydrophilic) at term

Imbibes water

Cervix becomes soft

Destabilizes collagen

Ripening

Other Substances Involved in Labour

- Relaxin

i. Towards term increases PG synthesis

ii. Activates collagenases

- VIP → increases contractions

- CRH
- Adrenergic stimulation
 - i. Alpha1, Alpha 2 favour contraction
 - ii. Beta 1 & Beta 2 → increases Amp
- Surfactant protein A
- Gastrin releasing polypeptide
- Placenta
- ↓
- CRH
- ↓
- Stimulates foetal HPO axis
- ↓
- Increases cortisol □ favours contraction
- ↓
- Decreases progesterone

Surfactant Protein A

- Produced by foetal lung
- Expressed by amnion /decidua
- Inhibits $PGF_{2\alpha}$
- Towards term → fall in surfactant protein A

Gastrin Releasing Polypeptide

- Smooth muscle stimulant

Favour Uterine Contraction

- Endothelin

- Platelet activating factor

CAPs: Contraction Associated Protein

- Oxytocin receptors
- Prostaglandin F receptors
- PG synthase
- Connexin 43



MECHANISM OF LABOUR

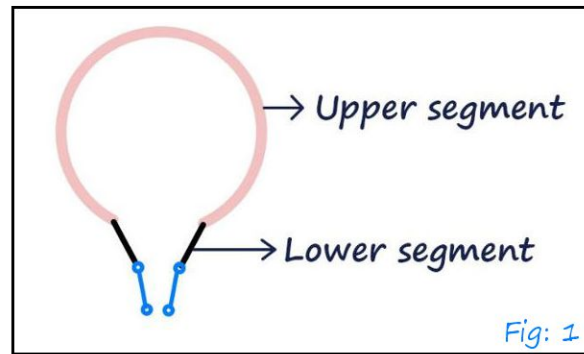
Definition: -

- Labour is the process by which the fetus after the period of viability is expelled from the genital tract
- Before the period of viability if the fetus is expelled
→ abortion
- Criteria to be met to classify labour as normal
 - Spontaneous in onset
 - Term gestation 37-42 weeks
 - Vertex presentation
 - Uncomplicated
 - Natural expulsive forces
 - Vaginal delivery

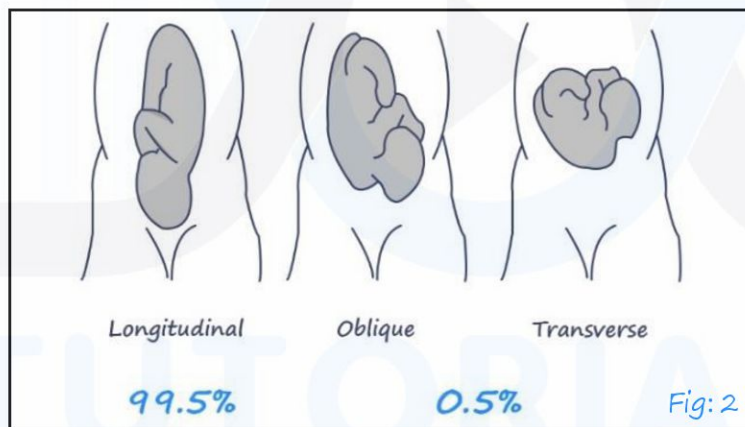
3 P's → passage → maternal pelvis
 passenger → fetus
 power → uterine contraction

Uterine changes: -

- Formation of upper uterine segment
 - Shortening of muscle – fibres
 - Thickening of uterine segment
- Formation of lower uterine segment
 - Stretching of muscle fibres
 - Thinning of uterine segment



- **Lie:** - Lie refers to the relationship of the long axis of the fetus to the long axis of the centralized uterus or maternal spine
- 3 types: Longitudinal oblique transverse lie

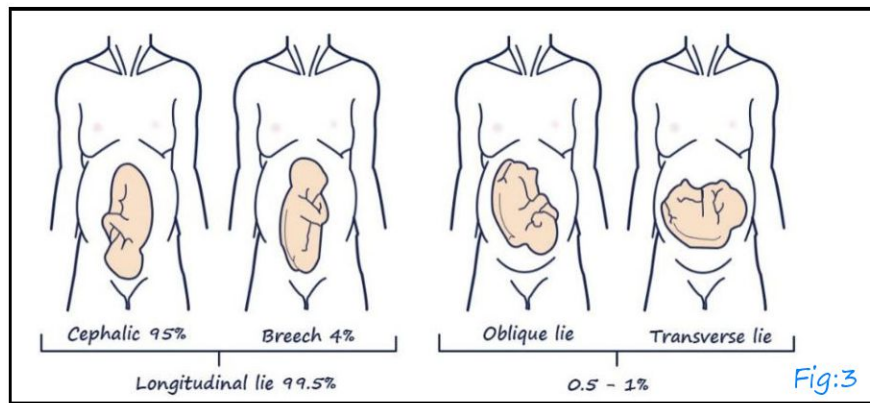


- **Presentation:** - The part of the fetus which occupies the lower pole of uterus

Cephalic - 96.5, podalic - 3%, shoulder - 0.5%

┌──────────┐
│
Seen in longitudinal lie

↓
Transverse lie

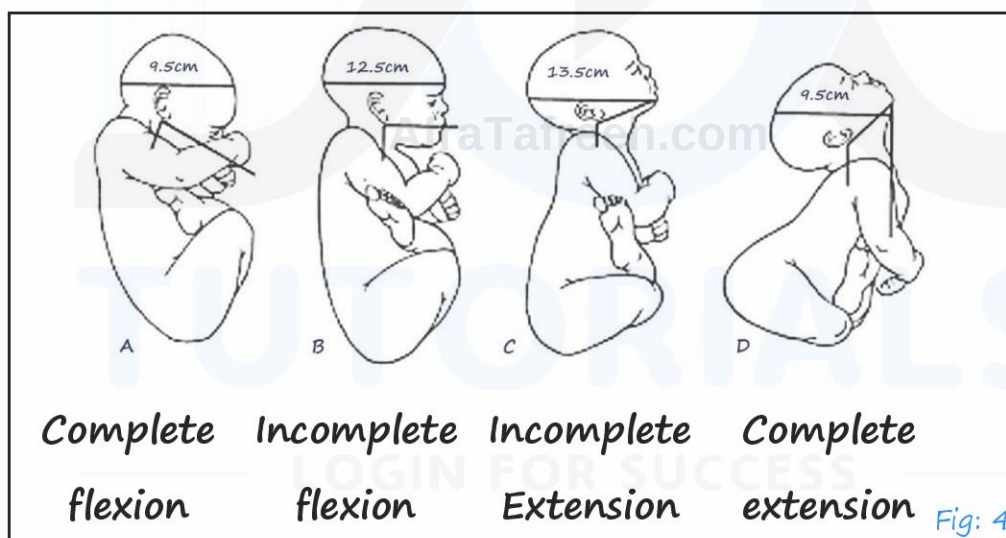


- Attitude: - Relationship of different parts of the fetus to one another

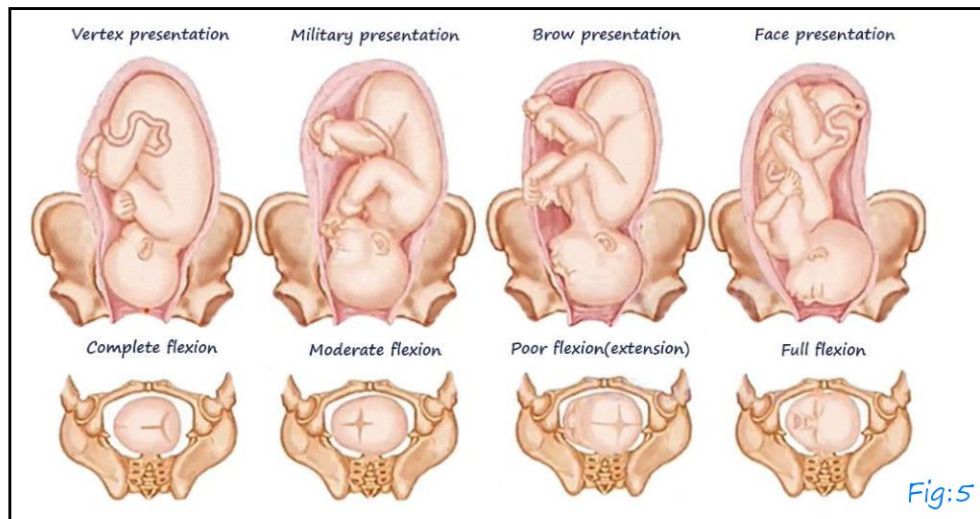
1. Flexion

2. Deflexion → incomplete extension

3. Extension



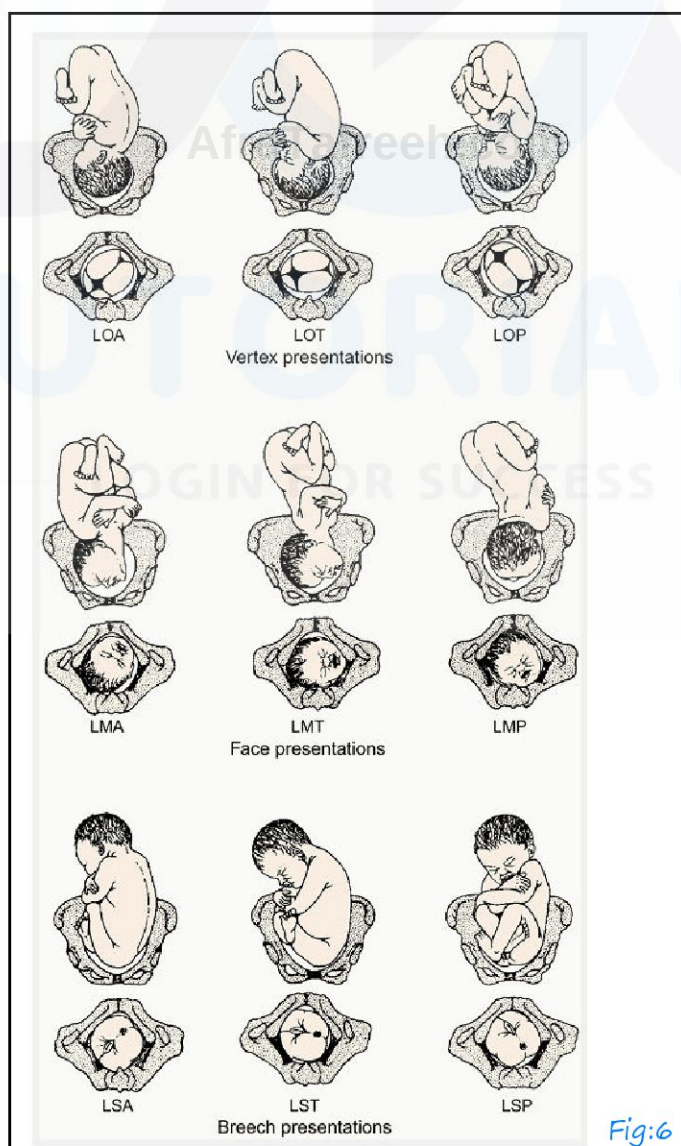
- Presenting part: - is defined as the part of the presentation which overlies the internal OS and is first felt on per vaginal examination



- Posterior fontanelle is felt (triangular in shape) → flexion / Incomplete flexion
- Anterior fontanelle is felt (Diamond in shape) → Deflexion
- In complete flexion, can neither feel anterior fontanelle nor posterior fontanelle
- Denominator: - It is an arbitrary bony fixed point on the presenting part which comes in relation with various quadrants of maternal pelvis
 - If attitude is deflexion → presenting part is vertex
Denominator is occiput
 - If attitude is deflexion or partial extension → presenting part → brow
Denominator → Frontal eminence
 - Complete extension → presenting part - face
Denominator - chin

Presentation	Attitude	Presenting part	Denominator
Cephalic	Complete flexion Deflexion Extension	Vertex Brow Face	Occiput Frontal eminence Chin
Breech			Sacrum
Shoulder			Acromion

- **Position:** - It is the relationship of the denominator to different quadrants of the pelvis



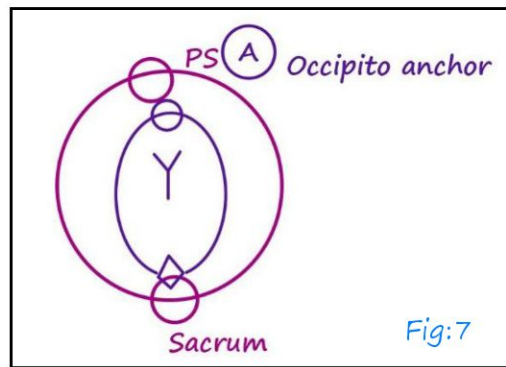


Fig:7

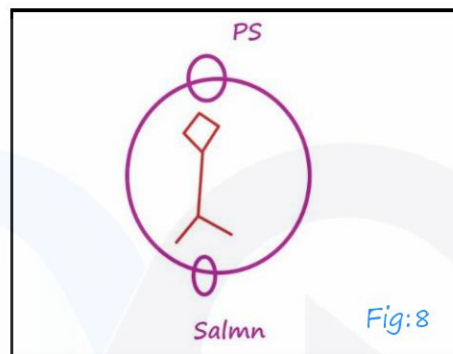
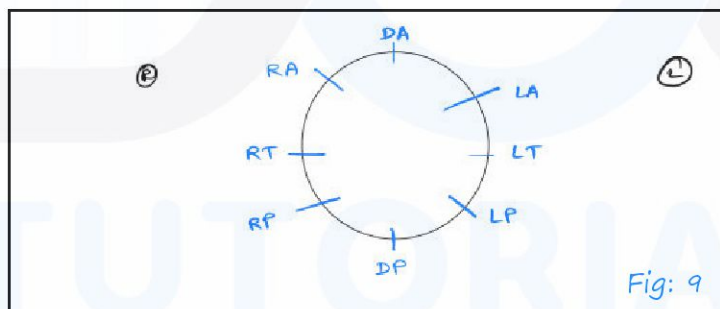
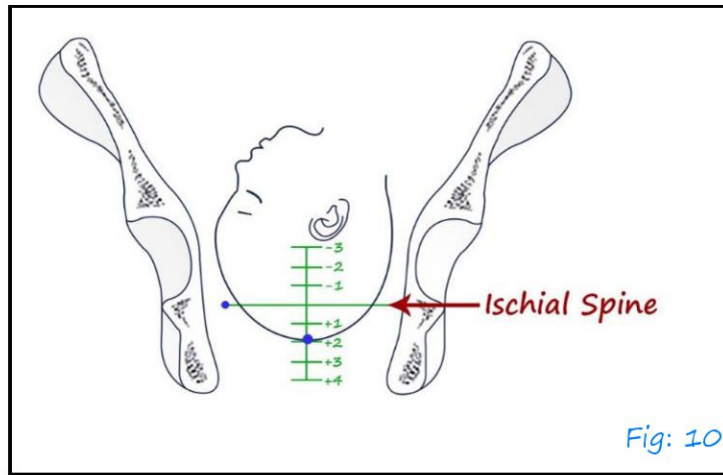


Fig:8



- Depending on where occiput sits, 8 different positions

Station: - refers to leading point of the skull in relation to ischial spine



- If leading point usually vertex is below ischial spine

1 cm → ++

2 cm → +2

3 cm → +3

Above ischial spine

1 cm → -1

2 cm → -2

3 cm → -3

At the level of ischial spine 0

- If the leading point of skull (vertex) touches the ischial spine – engaged
- Crichton method: – Abdominal method of assessing the descent of foetus

Abdominal Method (Crichton's Method)					
Completely above	Sinciput +++ occiput ++	Sinciput +++ occiput ++	Sinciput + occiput just felt	Sinciput + occiput not felt	None of head palpable
5/5	4/5	3/5	2/5	1/5	0/5
					
Level of pelvic brim					
'Floating' above the brim	'Fixing'	Not engaged	Just engaged	Engaged	Deeply engaged

Fig: 11

- 5/5 palpable → floating head / mobile head

Engaged

- P/A → Head is 1/5 or 0/5 palpable
- P/V → Vertex is at the level of ischial spine

Most common

- Lie → Longitudinal
- Presentation → cephalic
- Attitude → Flexion
- Position → L occipito transverse
- Engaging diameter → diameter of skull

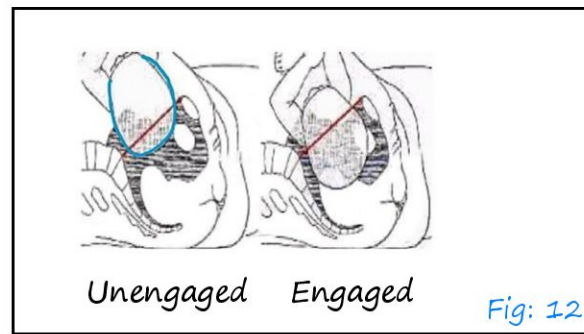


Biparietal diameter

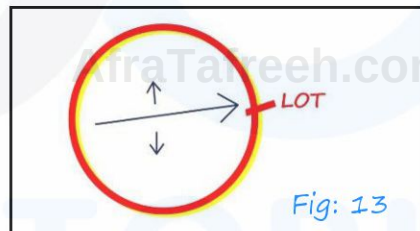
- Denominator → occiput

- Engagement: - Head is said to be engaged when the largest transverse diameter of the fetal skull crosses the brim of pelvis

- Largest transverse diameter → biparietal diameter



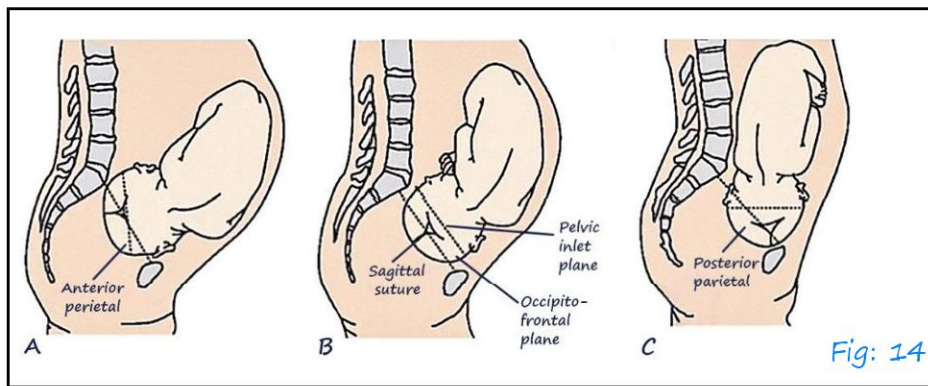
- Diameter of engagement → Diameter of pelvis which accommodates the sagittal suture of skull
- In lot position, diameter of engagement is transverse diameter of inlet
- LOA Position → R oblique diameter of pelvis
- Asynclitism:



- In lot position, if the sagittal suture moves anteriorly or moves posteriorly → Asynclitism



Deflection of sagittal suture towards pubic symphysis or sacral promontory



A, Anterior asynclitism. B, Normal synclitism. C, Posterior asynclitism.

Anterior asynclitism → sagittal suture moves backwards/posteriorly

Posterior asynclitism → sagittal suture moves anteriorly

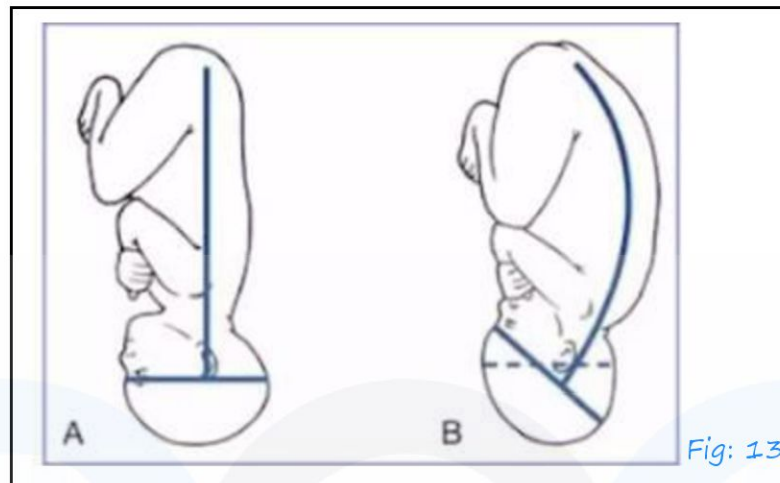
Cardinal movements

- Engagement
- Descent
- Flexion
- Internal rotation
- Extension
- Restitution
- External rotation

1. Engagement

2. Descent: - Downward movement of the foetus due to uterine contractions assessed by abdominal examination assessed by crichton method

3. Flexion: - Converts occipitofrontal (11.5 cm) incomplete flexion to suboccipito bregmatic (complete flexion) (9.5 cm)



- Flexion is brought about by resistance offered by
- Cervix (internal OS)
- Pelvic wall (Ischial spine) (most X)
- Pelvic floor (Levator ani)
- All these 3 are at same level.

4. Internal rotation

- Brings the occiput towards public symphysis
- Aligns SOB to AP diameter of pelvis
- Rotation is by 45°
- Occurs at the level of Ischial spine
- Forces: - Shape of pelvis
 - Impetus given by ischial spine
 - Elastic recoil of L.ani (most imp)

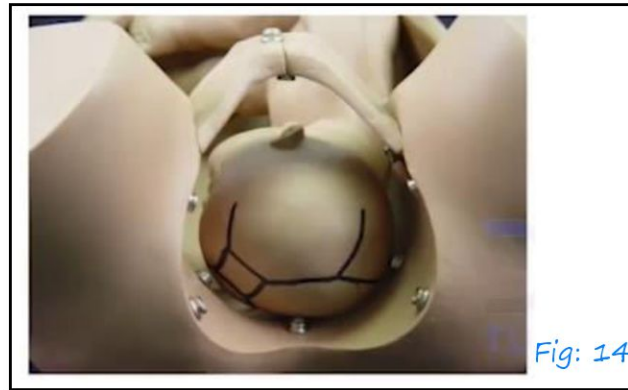


Fig: 14

Sagittal suture / SOB diameter

LOT Transverse diameter of pelvis

45° ↓



LOA

RO diameter

45° ↓



DOA

AP diameter

5.Extension: -

With further descent



Occiput hitches against the public symphysis



BPD stretches the vulval outlet



Pelvic canal curves anteriorly at this point,

Head extends gradually



Occiput, bregma, nose, mouth, chin delivered

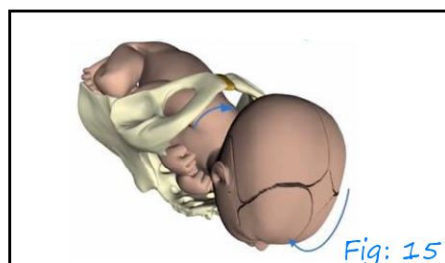
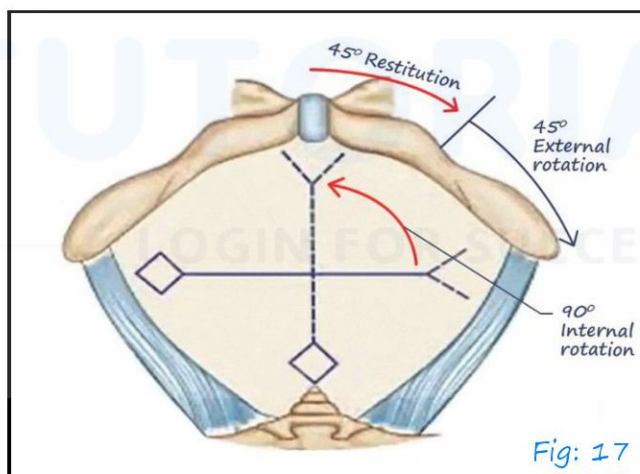
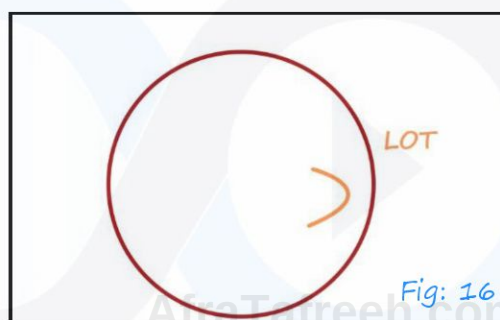


Fig: 15

- Forces: - 1. Shape of birth canal Q
- 2. Uterine contractions
- 3. Maternal expulsive efforts
- 4. Contraction Levator ani

6. Restitution: - untwisting of fetal neck first 45° → restitution

7. External rotation: - Brings shoulder from L.O diameter to AP diameter of pelvis 45° rotation
- Occurs in same direction as restitution



STAGES OF LABOUR & PARTOGRAM

Stages of labours

- Stage – I – Onset of true labour pain → full Cervical dilatation (10cm)
- Stage – II – Full cervix dilation → Delivery of baby
- Stage – III – Delivery of baby → Delivery of placenta
- Stage – IV – Delivery of placenta → 1hour observation after delivery of placenta

Labour Pains

00:03:03

True	False
Contractions at Regular intervals	Irregular
Increasing intensity of contractions	Doesn't not increase
Increasing Frequency of contractions	Doesn't not increase
Progressive cervical dilatation present	No dilation
Bag of membrane formation present	Absent
Show (blood mixed cervical mucus)	Absent

Normal duration of stages of labour 00:06:52

	Nulliparous	Multiparous
Stage 1	20 hours	14 hours
Stage 2	50 min	20 min
Stage 3		
• Without AMTSL	15 min 5 min	15 min 5 min
• With AMTSL		
Stage 4	1 hour	1 hour

AMTSL → Active Management of 3rd Stage of Labour

- Failure to progress: Lack of progressive cervix dilatation and fetal descent

Pressure changes

- Uterine baseline tone
 - Pre-labour → 8 mmHg
 - Labour → 12 mmHg
- Minimum pressure required to produce Palpable contractions – 10 mmHg

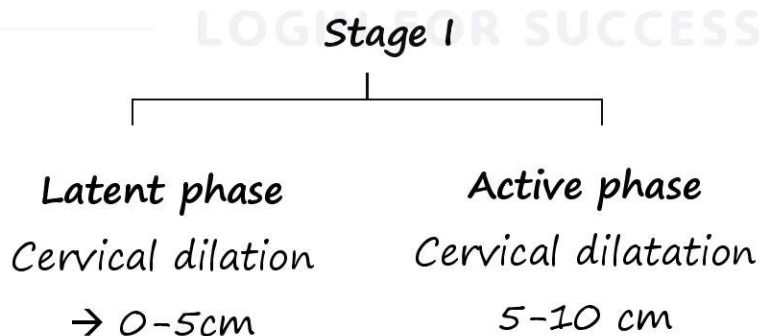
Hypotonic	Hypertonic
Basal tone is normal or decreased	Basal tone is high

Contraction – synchronous	Incoordinate uterine contraction
Pressure during contraction → inadequate	

- Minimum pressure required to cause pain 15 mmHg
- Minimum pressure required to dilate the Cervix 15 mmHg
- Pressure during normal spontaneous contraction – 60 mmHg (end of I stage, II stage)
- Pressure during commencement of stage I – 25 mmHg
- Pressure during end of stage I – 50 mmHg
- Pressure during second stage – 100 mmHg

Labour disorders

00:16:13



Rate of progression of labour

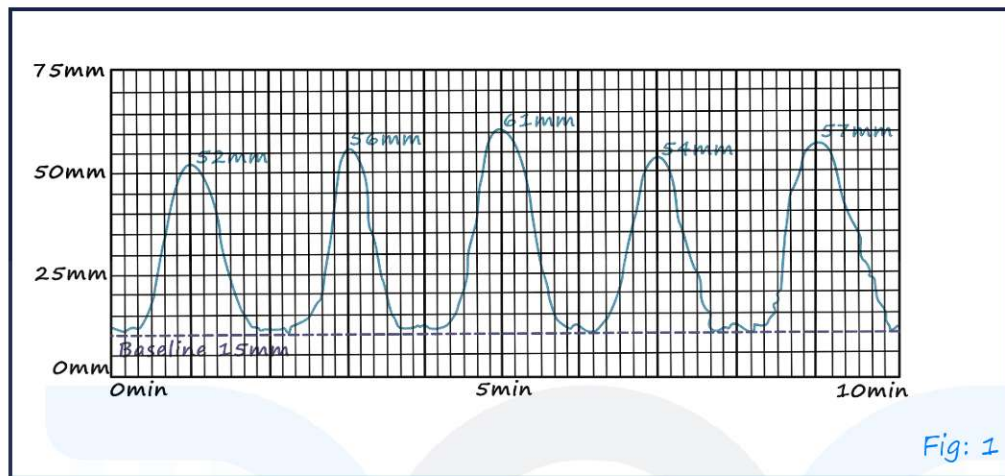
		Nulliparous	Multiparous
	Prolonged latent phase	> 20 hours	> 14 hours
Active phase	Protracted dilation	< 1.2 cm/hr	< 1.5 cm/hr
	Protracted descent (slow)	< 1cm/hr	< 2cm/hr

- Overall protracted dilation < 1cm/hr for 4 hrs
- Arrest disorders
 - Arrest of dilatation - No dilatation for > 2hrs
 - Arrest of Descent - No descent for > 1hr
- Adequate Uterine contraction
 - At least 3 contractions in 10 minutes each lasting for ≥ 40 sec

Inadequate Uterine Contraction

00:24:09

- Uterine contractions with a pressure of < 180 Montevideo unit (MVU)



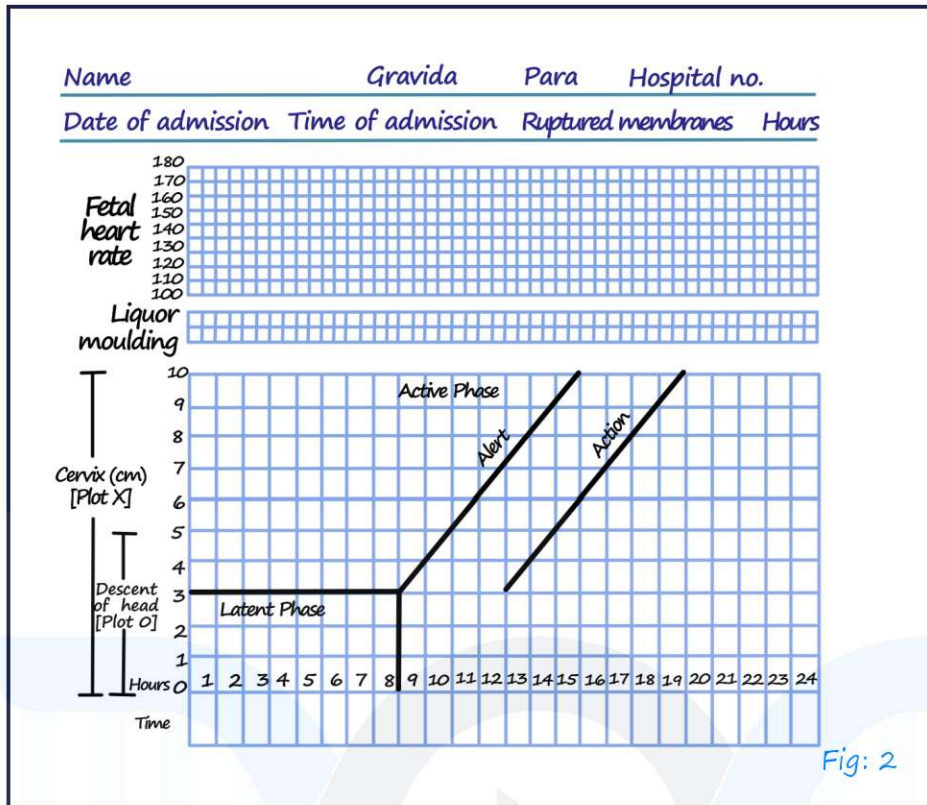
- Montevideo unit: Sum of pressure generated by each contraction over a 10 min period
- Baseline pressure $\rightarrow 15\text{mm}$
 - From the graph above:
 - Pressure during 1st contraction $\rightarrow 52 - 15 = 37$
 - Pressure during 2nd contraction $\rightarrow 56 - 15 = 41$
 - Pressure during 3rd contraction $\rightarrow 61 - 15 = 46$
 - Pressure during 4th contraction $\rightarrow 54 - 15 = 39$
 - Pressure during 5th contraction $\rightarrow 57 - 15 = 42$

205 Mv units

Partograph.

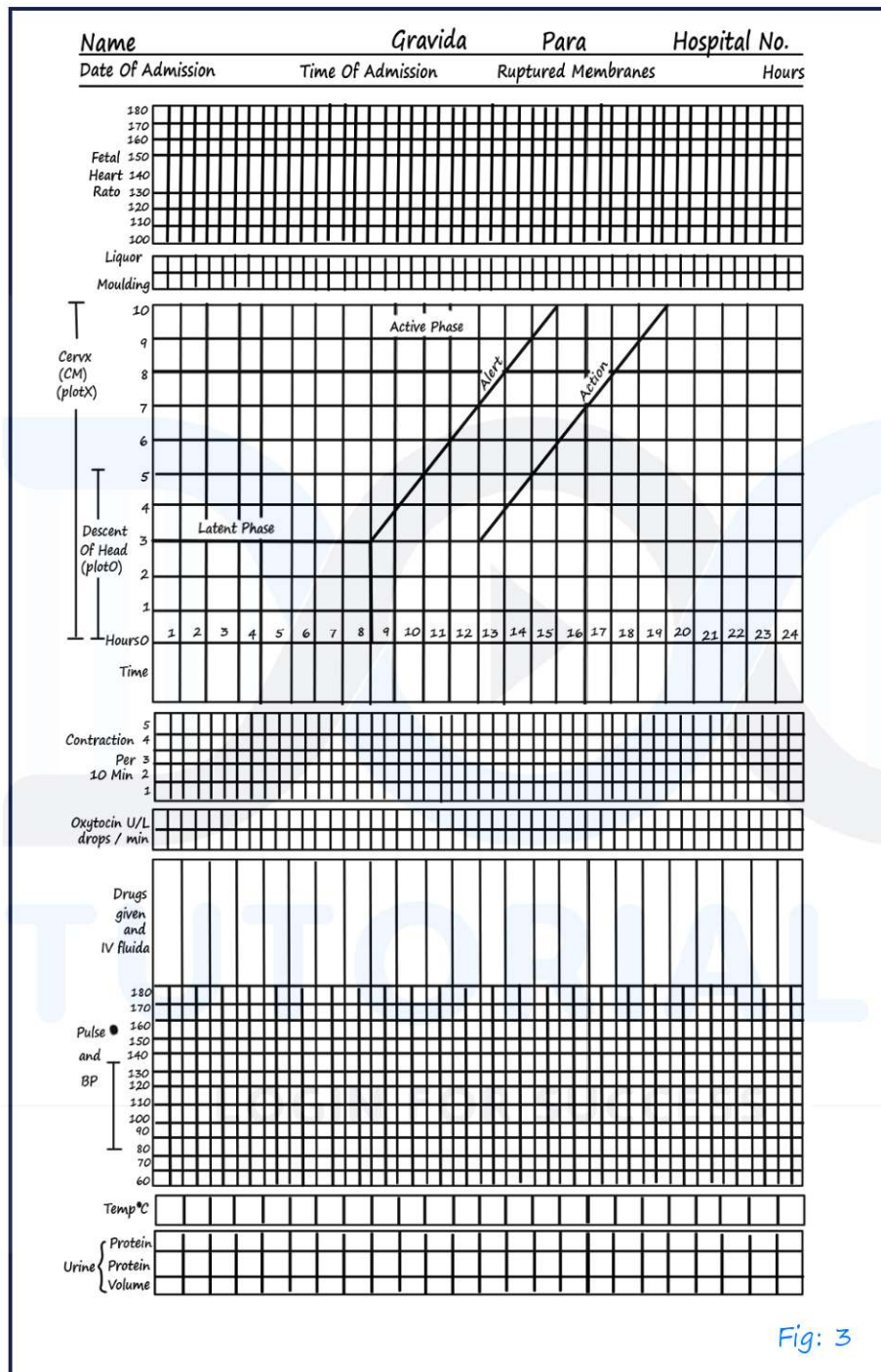
00:30:27

- According to new WHO guidelines,
- Partograph – plotted only in active phase



- FHR → Normal → 110-160
 - < 110 - Bradycardia
 - 160 → Tachycardia
- Liquor → C → Clear
 - I → Intact (membranes Intact)
 - M → Meconium Stained → **Fetal distress**
 - B → Blood stained → **Abruption**
- Moulding →
 - (+) → Parietal bones touch each other → **physiological**
 - (++) → Parietal bones override each other, reducible
 - (+++) → Parietal bones override each other, Irreducible

- Q Time gap $\rightarrow$ 4 hrs between Alert line and Action line



Dilatation

00:43:53

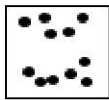
- 1 cm/hr → graph on the alert line – N
- >1cm/hr → graph → left of alert line – N
- < 1cm/hr → graph → Right of Alert line

or
Fall on action line
OR right of action line

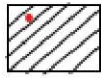
} Abnormal

- Primary dysfunctional labour → **most common dilatation abnormality seen clinically**
- Dilation is slow from beginning
 - From beginning → graph is to the Right of Alert line
- Most common cause → **poor contraction/Inadequate contraction**
- Secondary arrest of cervical dilation: is due to 3p's
 - P – power → uterine contraction
 - P – Passenger → Fetus
 - P – Passage → Pelvis

Contractions



< 20 sec



20-40 sec



> 40 sec - good contraction

- Most common cause for Primary dysfunctional labour → Inadequate contraction

Descent → represented by "0"

00:57:31

- By → per abdominal examination - **Crichton method**
 - 5/5 palpable → mobile head
 - 4/5 palpable
 - 3/5 palpable
 - 2/5 palpable
 - 1/5 palpable
 - 0/5 palpable
- fixed head/unengaged
- Engaged head
- Only when head engaged → Forceps/Vacuum can be applied

Prerequisites for Instrumental Delivery

01:00:58

- i. Fully dilated cervix Letter "X" – at 10cm
- ii. Fully descent/Engaged 1/5, 0/5 palpable Letter "O" at bottom line
- iii. Absent membranes –✱
- iv. No CPD □ 1+ 2+ ~~3+~~



INDUCTION AND AUGMENTATION

Induction

- Initiation of uterine contractions in a quiescent (silent) uterus.

Augmentation

- Acceleration of uterine contractions in a person in labour.

Indications for induction

Maternal	Fetal
- Gestational HTN - Preeclampsia - diabetes - Oligohydramnios - PROM	- IUD - IUGR - Post Dated pregnancy - Rh incompatibility. - Major fetal anomalies. - Non –reassuring fetal Heart rate

Clinical features

- Macrosomia
- CPD major
- Contracted pelvis
- Malpresentation

- Previous classical C-section
- Placenta Previa
- Hydrocephalus
- Grand multipara
- Prematurity
- Multiple pregnancy

Risk of induction

- Increased chances of cesarean delivery.
- Chorioamnionitis
- Uterine rupture
- PPH from uterine atony.

Bishop's Scoring

AfraTafreeh.com

00:05:35

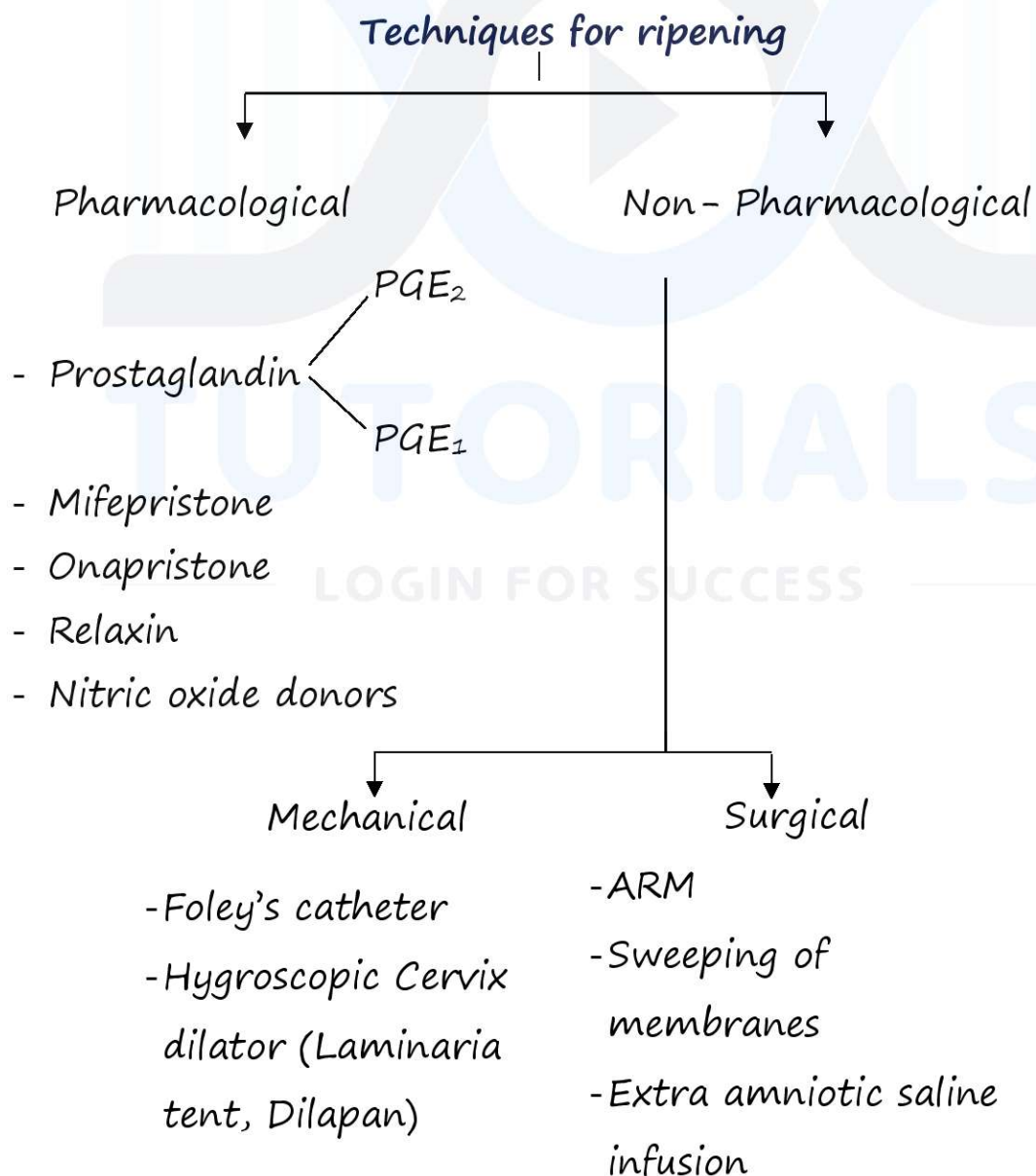
- Induction → means initiation of uterine contractions.
- Induction is successful only if the cervix is ripe.
Therefore, pre induction cervical ripening is done.

Bishop's scoring	0	1	2	3
D - Dilatation of Cervix	Closed	1-2cm	3-4cm	≥ 5 cm
E - Effacement of cervix	0-30%	40-50%	60-70%	≥ 80%

S - Station of head	-3	-2	-1, 0	+1, +2
C - Consistency of Cervix	Firm	Medium	Soft	
O - Position of Os	Posterior	Mid-position	Anterior	

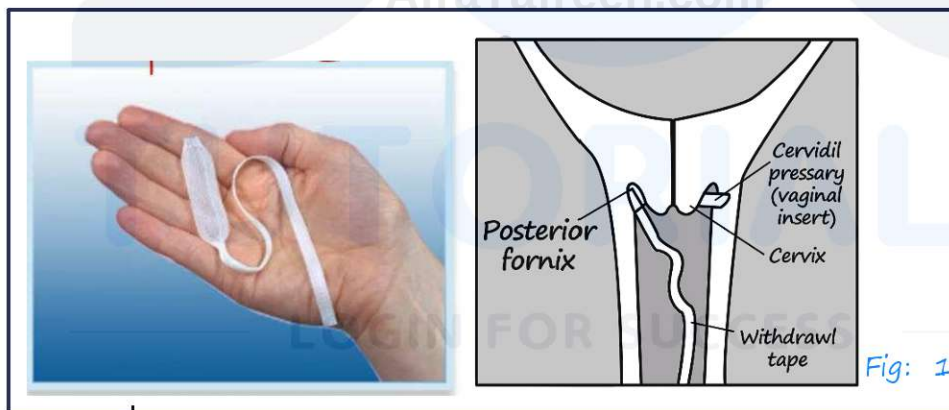
Max. score = 13

≥ 9 - Favorable



Drug	Preparation	Route	Dose
PGE ₂ (Dinoprostone)	Gel Vaginal insert Vaginal suppository	Intracervical Post Fornix Post Fornix	0.5mg 10mg 20mg → MTP between 12-20 weeks
PGE ₁ Misoprostol	Tablet 100mg/ 200mg	Vaginal/ Oral Oral/PR P/V	25mcg 50-100mcg 1000mg → PPH 800mg → MTP

- Mifepristone (RU 486)-200mg P/V x 2days
- Onapristone (ZK 98299)



- Vaginal insert- leave for 10-12hrs, remove 30min before starting oxytocin

PGE₂



Fig: 2

- Prefilled PGE₂ gel (Dinoprostone gel)
 - Intracervical gel, 0.5mg
 - max 3 doses at an interval of 6 hrs.

Kocher's forceps/ ARM forceps- used to rupture membranes

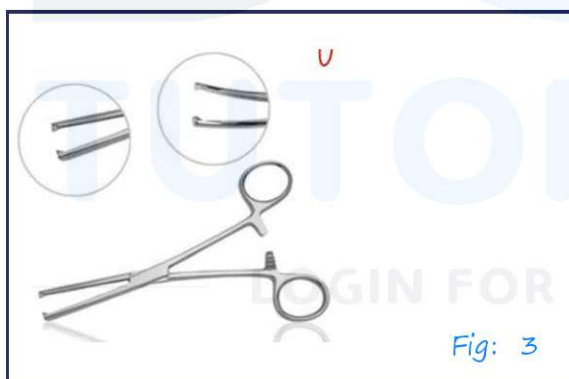


Fig: 3

Extra amniotic saline infusion-

- Foley's catheter is inserted & saline is infused.
- Saline goes between the decidua & the membranes
- This leads to the release of Prostaglandins.

Techniques for Augmentation

- Oxytocin

Not useful for unripe Cervix

Used for induction only in the ripe cervix.

- Whatever we use for induction of labour can also be used for Augmentation.

Oxytocin

- 10 units in 1000ml of RL
- 5 Units in 500ml of RL = 5000milli IU

$$1 \text{ ML} = 10\text{milliIU}$$
 - $t \frac{1}{2} \rightarrow 3 \text{ to } 5 \text{ mins}^Q$
 - Steady state is achieved in 30-40 mins^Q
- Duration of Action – 20 mins^Q
- ^Q Low dose oxytocin $\rightarrow$ starting dose 0.5 - 1.5milliIU/min
- ^Q High dose oxytocin $\rightarrow$ 4milliIU /min.
- Repeat 15-30 mins.
- Max dose -72milliIU/min, can be used without any compromise.

Good contraction

- Lasts for >40 second each, at least 3 contractions in 10 mins
- > 200 montevideo units.

Hazards of Oxytocin

- i. Tachy-systole: >5 contractions in 10 mins^Q
>7 contractions in 15 mins
- ii. Water Intoxication
 - Due to ADH like action -Decrease in free water clearance leading to Water intoxication
 - Convulsions /coma

Hazards of ARM

- Cord prolapse
- Infection
- Oligohydramnios
- Abruptio placenta

Questions

- 1Q: All are components of Bishop's score except.
 - a. Effacement of Cervix
 - b. Type of pelvis
 - c. Dilation of Cervix
 - d. Position of Os

Answer: B

- 2Q: Not an indication of labour induction.
 - a. Preeclampsia
 - b. Placenta Previa
 - c. Heart disease
 - d. Diabetes.

Answer: B

- 3Q: ARM is Contraindicated in
 - a. Hydramnios
 - b. Abruptio
 - c. Multiple Pregnancy
 - d. Heart disease.

Answer: A

- 4Q: Best Method of induction of Labour in hydramnios is
 - a. High rupture of membrane
 - b. Low rupture of membrane
 - c. Abdominal amniocentesis followed by stabilizing oxytocin drip
 - d. Prostaglandin.

Answer: C

- 5Q: Drug not used in Cervical ripening is
 - a. PGE_1
 - b. PGE_2
 - c. Mifepristone
 - d. PGE_{α}

Answer: D, PGE_2 α acts on myometrium

- 6Q: Induction is likely to be successful with a Bishop's score.
 - a. ≥ 4
 - b. ≥ 6
 - c. ≥ 8
 - d. ≥ 9

Answer: D

- 7Q: G3P2L2 @ 39 weeks with spontaneous labour pain comes with cervix 2cm dilated. 50% effaced, medium consistency, anterior in position with station @ $\square$ 2. Bishop score is
 - a. 4
 - b. 5
 - c. 6
 - d. 7

Answer: C

- 8Q: All of the following are advantages of PG over Oxytocin except
 - a. Oral administration
 - b. Better for women with poor Bishop score
 - c. Leads to water intoxication
 - d. Safe for women with heart disease.

Answer: C

- 9Q: ZK 98299 is
 - a. Mifepristone
 - b. Onapristone
 - c. Ulipristal
 - d. Asoprisnil

Answer: B

- 10Q: Tachysystole is
 - a. > 3 Contractions in 5 mins
 - b. > 5 Contractions in 5 mins
 - c. > 5 Contractions in 10 mins
 - d. > 7 Contractions in 10 mins

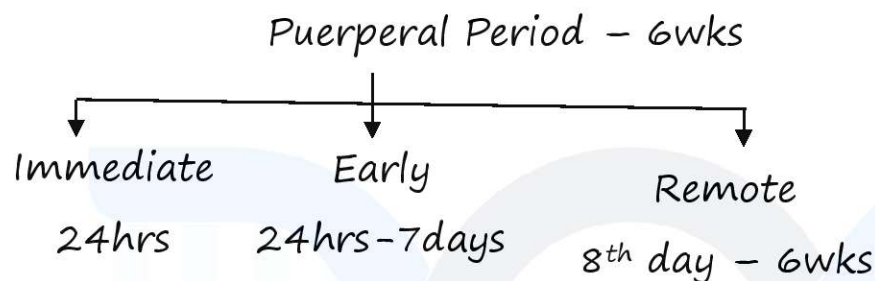
Answer: C

NORMAL PUERPERIUM

Definition

00:00:05

- Period after childbirth during which the maternal tissues return back to the pre pregnant state
- Usual time taken – 6wks



Uterus

Changes	Weight
Immediate postpartum	1000 grams
After 6wks	60grams Weight of nonpregnant uterus

- Placental site immediate postpartum → raised to 7.5cm
- After 6wks → 1.5cm
- Pregnancy → uterus undergo hypertrophy with limited hyperplasia
- In Puerperium → size only decreases not the number of cells

Endometrium

- Sheds with delivery
- Regeneration starts by day 7 postpartum
- Regeneration completes by day 10 post-partum
- Endometrium is completely restored by day 16
- **Endometrium is completely restored at placental site – 6wks**

Involution

- Shrinking of uterus to its pre pregnant state
- Rate of involution – 1.25cm/day
- At the end of 2wks post-partum- Uterus become a pelvic organ
- Complete involution = 6wks post-partum

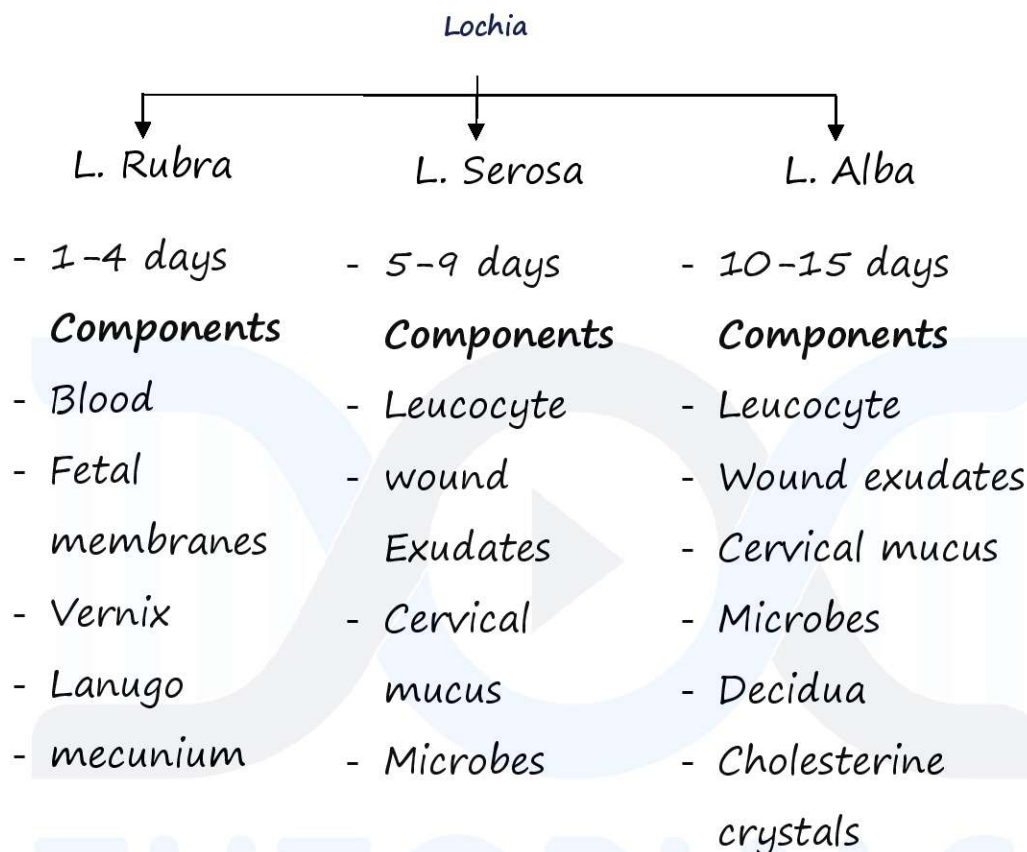
Vagina

- Pregnancy is High estrogen and progesterone state
- Good nourishment of vaginal epithelial cells, all 4 layers superficial layer, intermediate layer,
- Para basal layer and Basal layer –well developed
- Vagina completely involutes by 6-10wks
- Hymen-Lacerated during pregnancy replaced by nodular tags □ **caruncle myrtiformes**

Lochia

- Vaginal discharge for the first 2wks post-partum and has offensive fishy odour

- It includes shedding of endometrium, bleeding from genital tract, fetal remnants like, Vernix, lanugo, amniotic fluid, some microbes
- Normal amount of lochia = 250ml/day



General Changes

1. Pulse rate

- Transient increase, which returns to normal on day 2

2. Temperature rates

- Increases by 0.5°F → return to normal within 12hrs

3. Urinary tract

- Bladder congested/urinary retention
- Diuresis □ day 2, day 3 - More prominent in pre-eclampsia

- Ureter/renal pelvis $\square$ return to normal by 8wks post-partum

4. GI tract - Increased thirst and Constipation

Hematological Changes

- Blood volume $\rightarrow$ During pregnancy – increases by 30-40%, peak at 32wks GA
- Returns to normal by 2wks post-partum
- Cardiac output $\square$ returns to normal by 1wk post-partum
- RBC volume/hematocrit returns to normal by 8wk post-partum
- Leucocytes: increase up to 25000 post-partum

Clotting System

- Platelet
 - Pregnancy – Decreases
 - Puerperium – Increases upto 4-10 days
- Fibrinogen
 - Pregnancy – Increases
 - Increases upto 2wks puerperium
 - Postpartum period-Hyper coagulable state $\rightarrow$ high risk of DVT
 - Fibrinolytic System
 - Pregnancy: Depressed
 - Puerperium: Increased – to prevent clot formation

	<i>Pregnancy</i>	<i>Puerperium</i>
<i>Platelets</i>	<i>Decreases</i>	<i>Increases</i>
<i>Fibrinogen</i>	<i>Increases</i>	<i>Increases</i>
<i>Fibrinolytic activity</i>	<i>Decreases</i>	<i>Increases</i>



ABNORMAL PUERPERIUM

Postpartum Depression

00:00:05

- Incidence: - 10-20%
- Risk factors:
 - Young age
 - Antenatal depression
 - Unmarried status
 - Smoking
 - Newborns requiring ICU
 - H/O stressors during pregnancy
 - Physical / verbal abuse during pregnancy
- Recurrence rate: - 70%
- Depression during the 1st week and month after delivery can lead to insecure attachment and later Behavioural problems in the child

Treatment

00:02:59

- Mild to moderate depression: - Behavioural therapy
- Moderate to severe depression: - antidepressants
 - 1st DOC: - SSRI
 - TCA, MAO inhibitors clinical in pregnancy
 - Q. Medication should at least be continued for 6 months
 - Q. Relapse rate on discontinuing treatment: - 70%

Dietary deficiencies

- Omega 3 fatty acids
- Iron
- Folate
- Riboflavin
- Calcium
- Vitamin D
- DHA

Side effects

- Antidepressants
 - Preterm birth
 - LBW (Low Birth weight)
- Q. Paroxetine: - Teratogenic → Cardiac defects → VSD
- Q. SSRIs after 20 weeks
Increases risk of pulmonary hypertension abrupt discontinuation of SSRI will lead to
 1. Withdrawal symptoms
 2. miscarriage
- ECT is only for major mood disorders unresponsive to pharmacotherapy Q

Complications of ECT

- Fetal arrhythmias
- Vaginal bleeding
- Abdominal pain

- Self-limiting contractions
- Aspiration
- Aortocaval compression
- Respiratory alkalosis

Condition	Timing	Clinical features	Treatment
Postpartum blues	Begins 2-3 days after delivery peaks by D5 subsides by 2 weeks	Mood swings anxiety crying spells	Supportive mild tranquilizers
Depression	2 weeks after delivery later than blues later for > 2 weeks	Insomnia low energy LOA / LOW anger feeling of inadequacy	Supportive antidepressants
Psychosis	Within 1 st month	Mania hallucination delusion psychotic behaviour	Hospitalize antipsychotic ECT

PRETERM LABOUR

Definition

00:00:22

- Onset of labour before 37 completed weeks

Types

00:00:54

1. Threatened → Only contractions are present
2. Established → There are cervical changes along with uterine contractions (effacement and dilatation)

Criteria

00:02:31

- Regular contractions
- If there are more than or equal to 4 contractions in 20 minutes (or) ≥ 8 contractions in 60 minutes
- Cervical effacement of $\geq 80\%$
- Cervical dilatation of > 1 cm

Classification

00:04:20

- Early preterm: - less than 34 weeks
- Late preterm: - 34 to <37 weeks
- Near term or early term: - 37 to <39 weeks
- Term: - ≥ 39 weeks

Causes / Risk Factor	
Uterine overdistension	Periodontal disease
Prior preterm	Smoking
Stress	Inadequate weight gain
Infection (Gardnerella, Mycoplasma)	Young and advanced age
Cx dysfunction	Poverty
Hydramnios	Short stature
Uterine anomalies	Vitamin C deficiency
Fetal anomalies	Depression/Anxiety
Gestational hypertension	Hard labour
Autoimmune diseases → SLE, APLA	

- Stress → increases cortisol level

Types of cerclage

00:10:09

- Cerclage – suture that is applied to the cervix
- 2 types
 - Trans vaginal cerclage
 1. Mc Donald (Most common)
 2. Shirodkar → most effective
 3. Wuems suture → Emergency
 - Transabdominal
 1. Laproscopic cerclage
 2. LASH
- Transvaginal procedures done only during pregnancy
- Transabdominal procedures done in pre-pregnant state. Delivery only by C-section
- Transvaginal cerclage should be removed at 37 completed weeks → vaginal delivery is feasible.

POST DATED PREGNANCY

Post-dated Pregnancy

00:00:30

- Definition

- Pregnancy exceeding 42 completed weeks or 294 days
- Incidence is 0.4%
- Establish the Gestational Age in the 1st trimester with Ultrasound

Causes:

- Obesity BMI > 25 - Nulliparity - Increased Cx length - Family history (maternal genes) - Anencephaly → reduced CRH - Adrenal Hypoplasia → reduced cortisol → X linked placental sulfatase deficiency → Oestrogen	Postmaturity Syndrome - Wrinkled, patchy peeling skin (especially in palms / soles) due to loss of vernix - Long thin body (wasting) - Open eyes - Alert / Old man look / worried look, long nails
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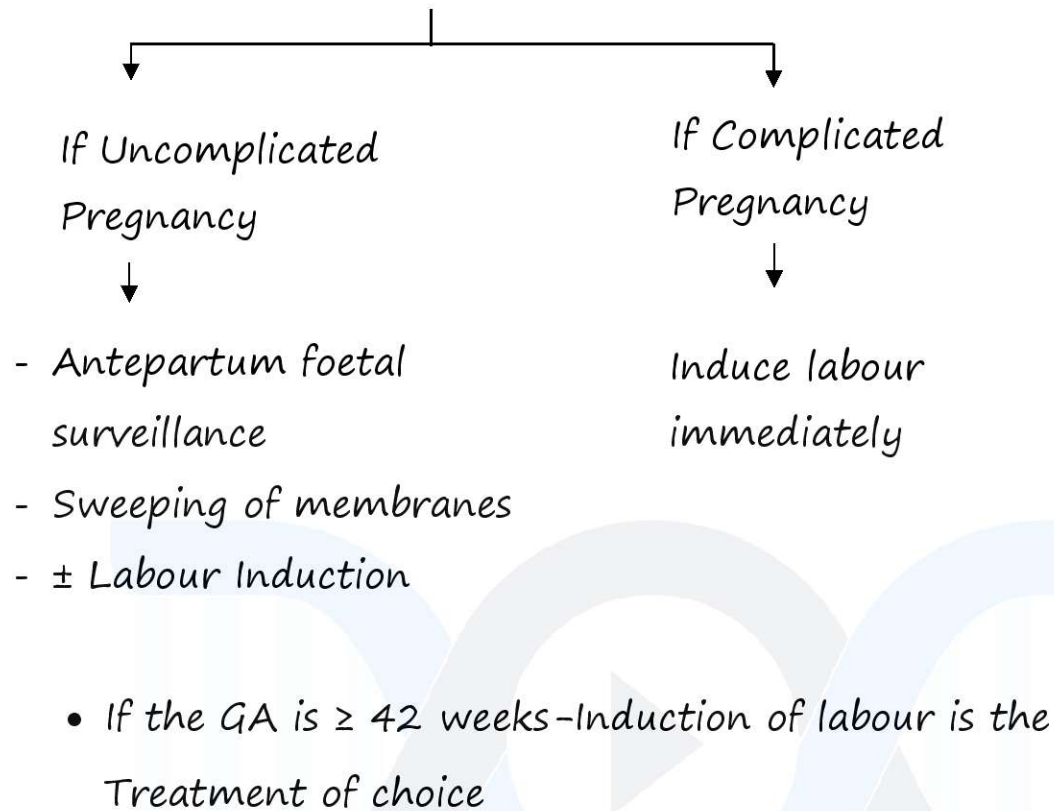
Pathology with advancing GA

- Increase in Kisspeptin (proapoptotic substance)
 - Placental dysfunction
 - Hypoxia
 - Increase in Erythropoietin levels (marker for postdatism)
- Adverse Maternal outcomes with post-dated pregnancy:

- Macrosomia - IUGR - Oligohydramnios - Preeclampsia - Increased rates of CS - Shoulder Dystocia - PPH	- Perineal Lacerations - Still birth - Post maturity syndrome - MAS - Neonatal convulsions - HIE - Birth injuries to the baby - Childhood obesity
--	--

Management

- If the GA is 41 – 42 weeks



TUTORIALS

LOGIN FOR SUCCESS

OCCIPITOPOSTERIOR POSITION

Definition

00:00:05

- Occiput is placed over sacroiliac joint or sacrum
- Lie: Longitudinal
- Presentation: Cephalic
- Presenting part: Vertex
- Attitude: Flexion

Types:

- i. Right occiput posterior ROP → Most common
- ii. Left occipito posterior
- iii. Direct occipito posterior

Causes:

1. Abnormal pelvis (Anthropoid pelvis)
2. Deflexed head
3. High pelvic inclination
4. Anterior placenta
5. Abnormal uterine contraction
6. Fetal anomalies (Brachycephaly)

Engaging diameter of skull

- Occipito frontal (or) sub occipito frontal

Diameter of engagement (Pelvis)

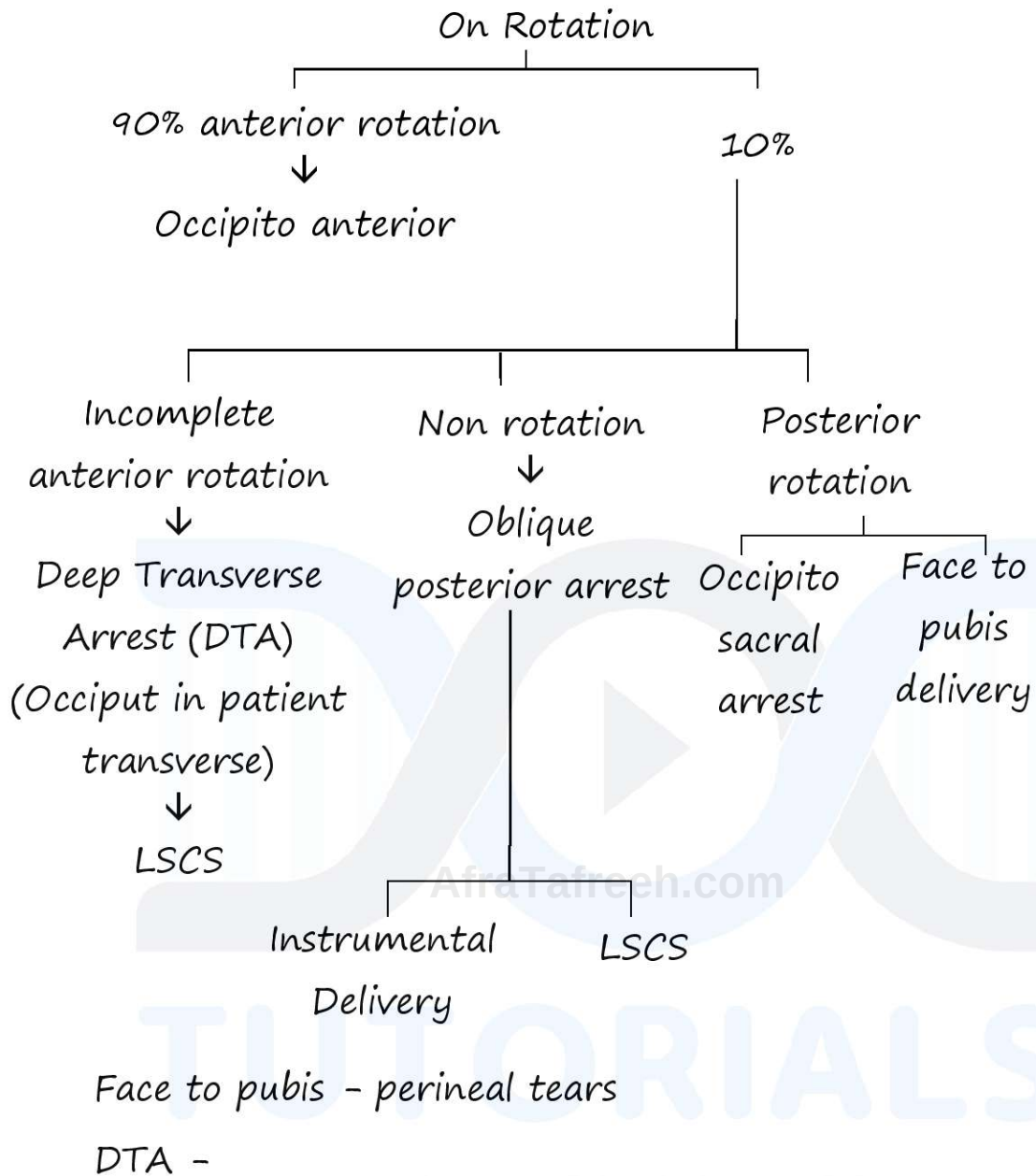
- i. In right oblique – ROP
- ii. In left oblique – LOP

Engaging diameter

SOB (Suboccipito bregmatica)	SOF (Sub occipito frontal Bregma)	OF (Occipito frontal)
Vertex presentation Occipito anterior Complete flexion 9.5 cm	Vertex presentation Occipito posterior Incomplete flexion 10.5 cm	V _x presentation Occipito posterior Incomplete flexion 11.5 cm

LOGIN FOR SUCCESS

Management



FACE AND BROW PRESENTATION

Face presentation

- Lie: Longitudinal
- Presentation: Cephalic
- Presenting part: Face
- Attitude: Extension
- Denominator: Mentum

Types of face presentation

- Right mento anterior
- Left mento anterior – most common
- Right mento posterior
- Left mento posterior

Causes of face presentation

00:03:42

Causes of similar to breech and face

- Intrauterine death
- Anencephaly (most common congenital anomaly with face presentation)
- Multiparity
- Pre maturity

Causes related to neck

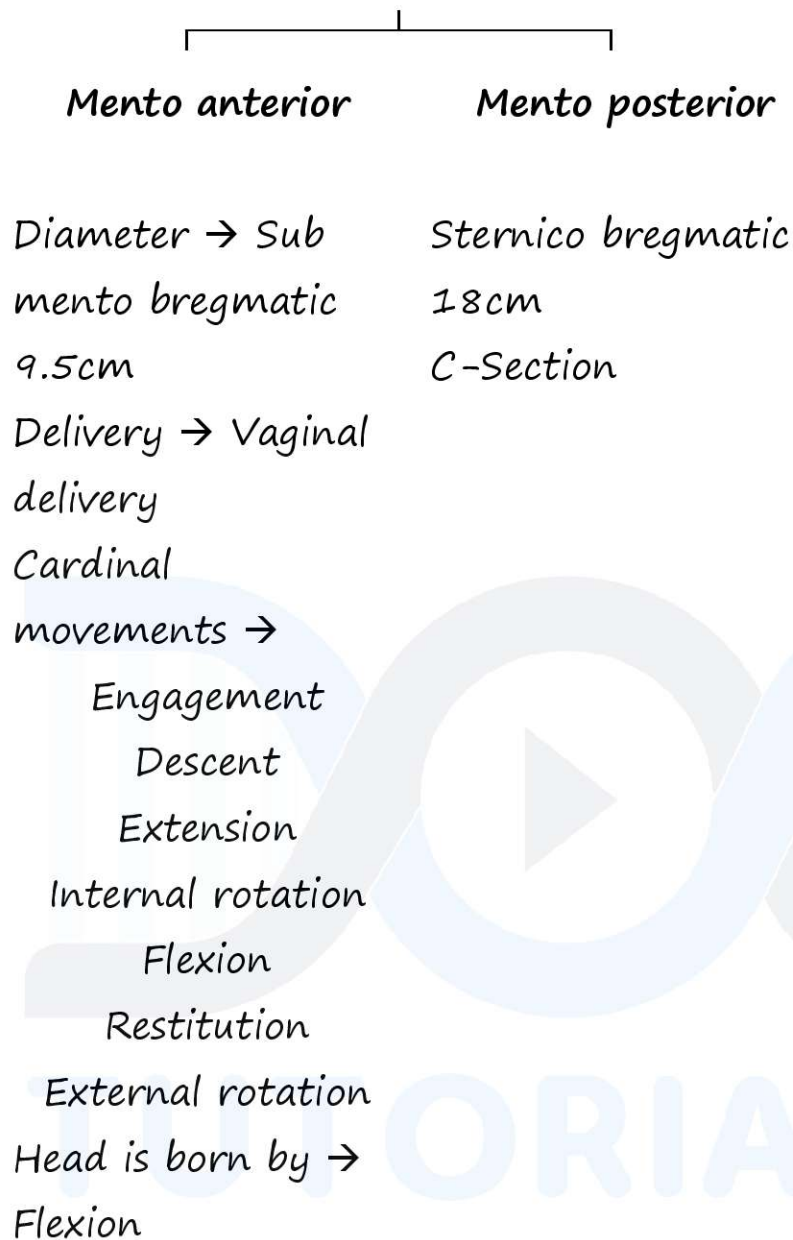
- Most common – anencephaly
- Congenital bronchiole, goitre, cystic hygroma
- Twist of cord around neck
- Increased group of extensor group of neck muscles

Other causes

- CPD, flat pelvis, lateral obliquity of uterus

	<i>Part-Felt</i>	<i>Attitude</i>	<i>Position</i>	<i>Diameter</i>
1	Only posterior fontanelle	Complete flexion	SOB	9.5 cm
2	Posterior and anterior fontanelle	Incomplete flexion	SOF OF	10.5 cm 11.5 cm
3	Only anterior fontanelle + Suboccipital ridge (Brow presentation)	Neutral position	Mento vertical	14 cm
4	Non fontanelle felt only facial parts (Face presentation)	Complete extension	Sub mento bregmatic	9.5 cm

Face presentation



Brow presentation

- Engaging diameter – 14 cm Mento vertical
- Attitude – Neutral position (Neither extension nor flexion)
- P/V – Supraorbital ridge/ Anterior fontanelle)
- Causes → are same as face presentation
- Most common causes → Flat pelvis (Platypelloid)
- Delivery – Always by LSCS.

BREECH PRESENTATION

Incidence

- @ 28 weeks ↑ 20%
- @ 34 weeks ↑ 5%
- @ Term ↑ 3%
- Most common of breech – Prematurity

Causes

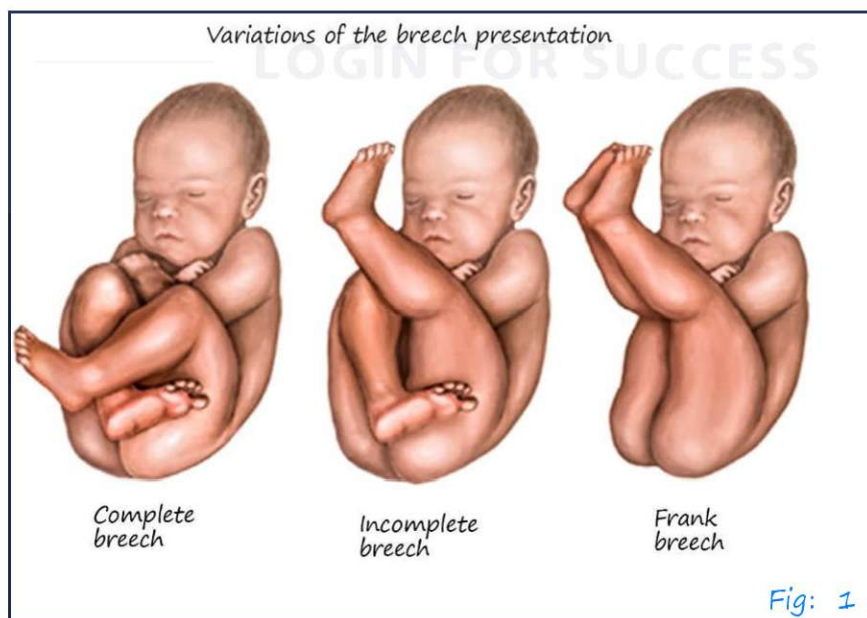
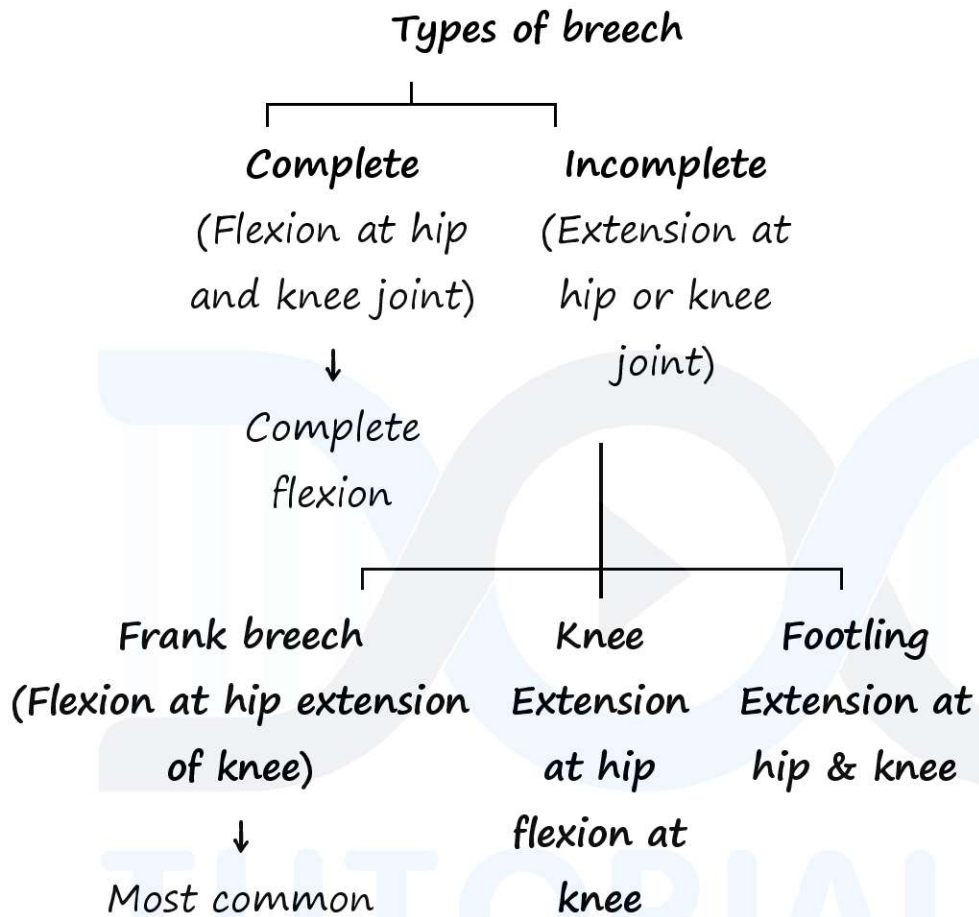
- Anomalies of uterus (Septate/ bicornuate)
- Anomalies of fetus (Trisomy 13, 18, 21)
- Short cord
- Intrauterine death
- Breech with extended legs
- Oligohydramnios
- Turns

Other causes

- Favourable adaptation
- Hydrocephalus
- Placenta previa
- Corner fundal attachment of placenta
- Cephalon Pelvic Disproportion (CPD)

Under mobility of fetus

- Hydramnios
- Multipara with lax abdomen



Breech

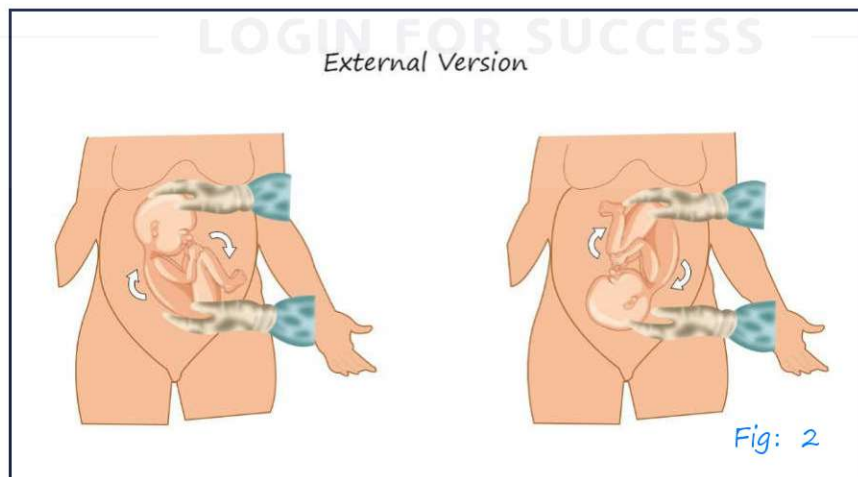


Diameter of engagement

- Breech → Bitrochanteric → 10cm
- Shoulder → Brisacromial → 12cm

External cephalic version

- Conversion of breech presentation → Cephalic





Contraindications of ECV (External cephalic version)

00:07:12

- Antepartum hemorrhage → P. Placenta previa/abruption
- IUD/IUGR/Macrosomia/Extended head
- Multiple pregnancy
- PROM
- Uterine anomalies/abnormal CTG
- Contracted pelvis/CPD
- Previous > 1 LSES/Elderly primi/Bad obstetric history (BOH)
- Precious pregnancy
- Complicated breech
- When to ECV → Ideal time – 36 weeks
- Tocolysis before ECV → To be given
- Anti-D (In case of Rh-Negative pregnancy) before ECV → to be given.
- Anesthesia → not required

Management of breech:

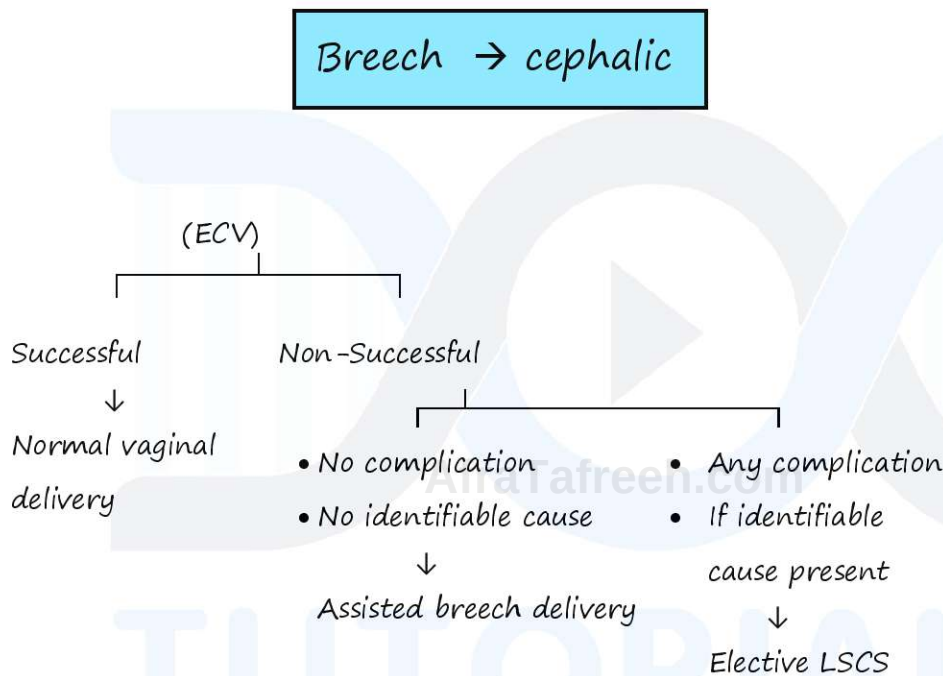
Breech



wait till 36 weeks



Therefore at 36 weeks external cephalic version can be done



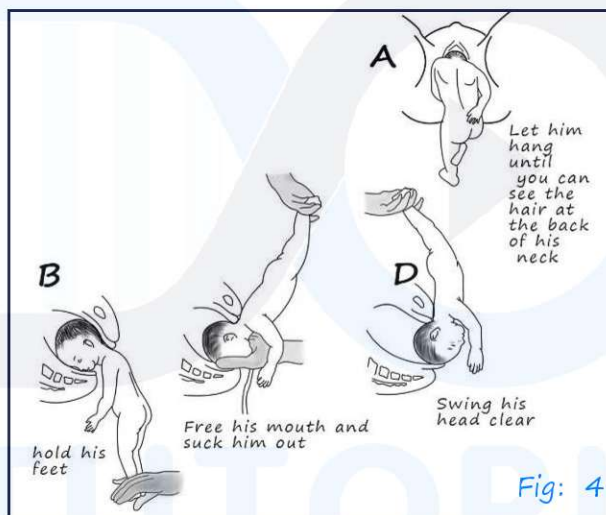
Indications for elective LSCS (> 38 weeks)

- Fetal weight → <1500 or >3500 gm
- All complicated pregnancies like APH, Oligohydramnios, fetal distress, CPD
- Hyperextended head → stargazing fetus
- Footling presentation
- Knee presentation
- Complete breech
- Previous LSCS.

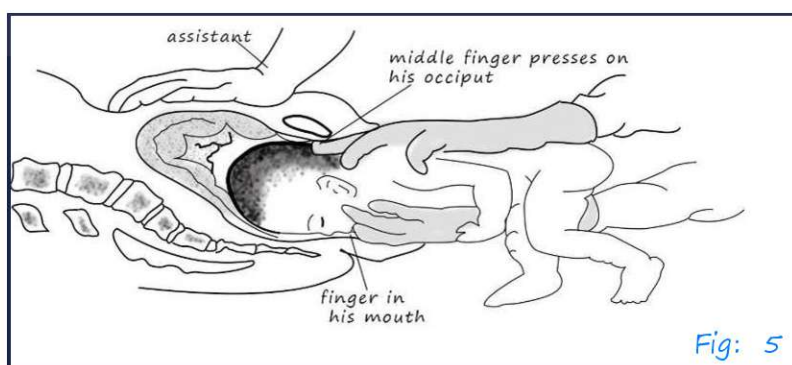
Assisted breech delivery:

00:11:28

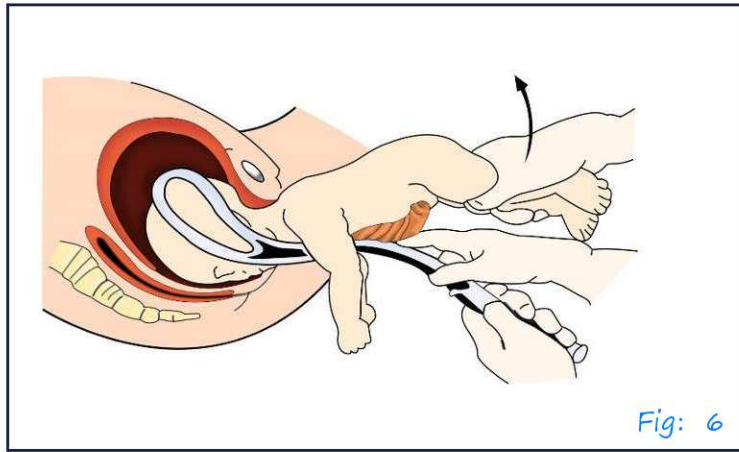
- Delivery of head
 - Burns marshall method
 - Mauriceau smellie veit method
 - Peper's forceps.
- Extended legs → Pinard's maneuver (Pinards – Popliteal fossa)
- Extended arm → Lovset maneuver
- Burn Marshall method



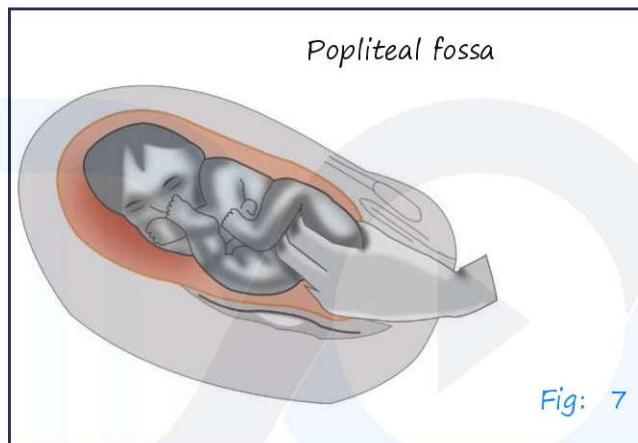
- Mauriceau smellie veit method



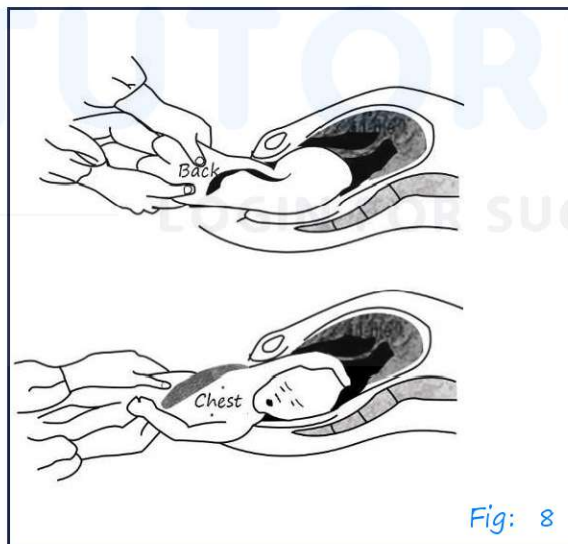
- Pipers forceps:



- Pinards:



- Lovset's



TRANSVERSE LIE, UNSTABLE LIE AND COMPOUND PRESENTATION

Transverse lie

00:00:14

Definition

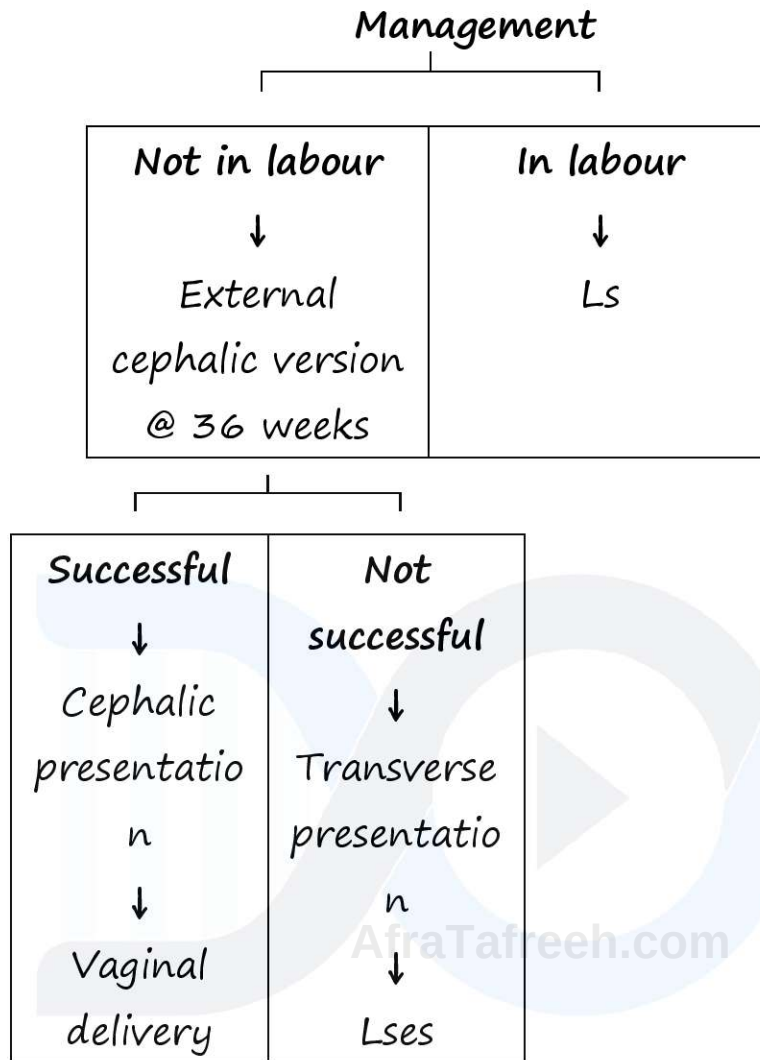
- Long axis of the fetus lies perpendicular to the maternal spine

Types

- Dorso anterior → most common
- Dorso posterior
- Dorso superior
- Dorso inferior

On examination

- Fundal height → Smaller for given gestation
- Fundal grip → Does not show breech/ head
- Lateral grip → One side breech, other side head
- Pelvic grip → Lower pole empty
- Presenting part: Shoulder
 - Denominator → Acromion process
- Complications:
 - Cord prolapses
 - Cord presentation
 - Hand prolapses
- *Conduplicato corpore* → Seen in transverse lie ^Q



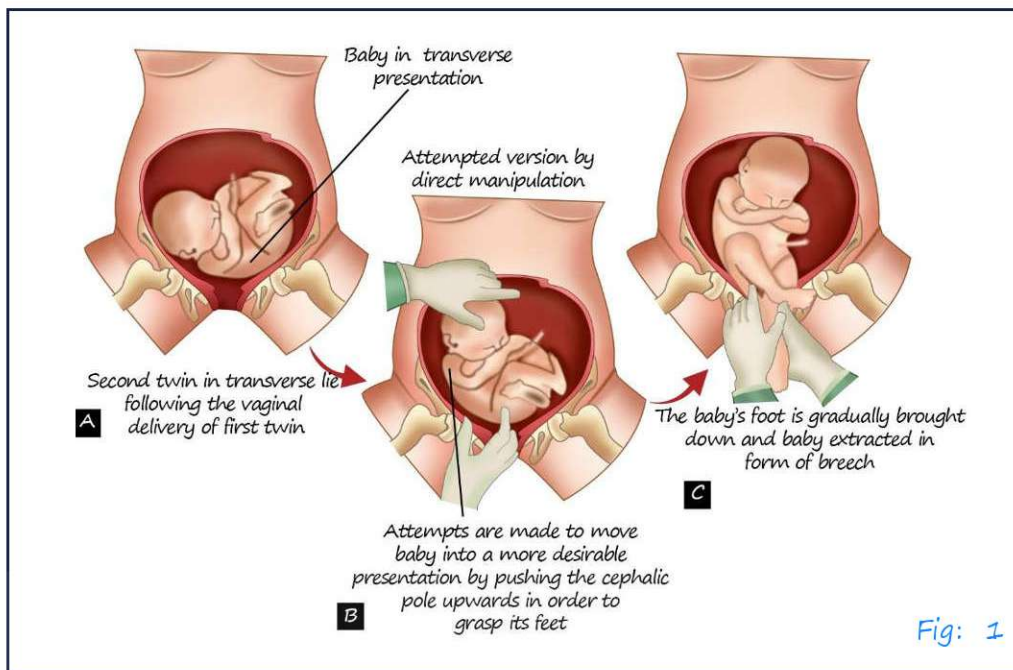
Internal podalic version (IPV)

00:08:25

- In modern obstetrics allowed only for 2nd twin where 1st twin → cephalic, 2nd twin → transverse lie

Transverse lie $\xrightarrow[\text{Concerted to}]{\text{IPV}}$ Breech

- Complication of IPV → Uterine Rupture^Q



Compound presentation

- Along with presenting part □ hand or foot coming down
- Most common type □ head with hand
 - Vaginal delivery

TUTORIALS

LOGIN FOR SUCCESS

CORD PROLAPSE

- Cord coming near the presenting part

Types

- Occult prolapse: Cord placed by the side of presenting part
- Cord presentation: Cord is below the presenting part with intact membranes
- Cord prolapse: Cord is below the presenting part with ruptured membranes

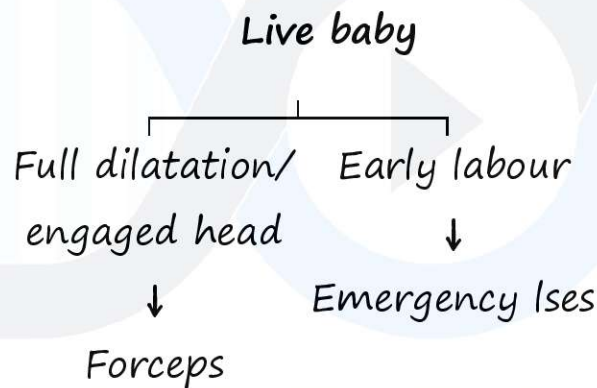


Causes

Transverse lie	Twins
Breech (footling)	Hydramnios
Contracted pelvis	Placenta previa
Prematurity	Rupture of membranes during ECV/ IPV

Management

- Cord presentation → Don't try to replace the cord
 - Do emergency lses
- Cord prolapses
 - Immediate repositioning
 - Stop oxytocin
 - Fill the bladder (to prevent cord from coming down)
 - Foot and elevation
 - Exaggerated Sims position



Dead baby

↓

Wait for vaginal delivery

PROLONGED LABOUR, OBSTRUCTED LABOUR,

PRECIPITATE LABOUR

Prolonged labour

00:0014

- Varies for nulliparous and multiparous

	Nulliparous	Multiparous
Prolonged latent phase	>20 hours	>14 hours
Protracted dilatation	<1.2 cm/hour	<1.5 cm/hour
Protracted descent	<1cm/hour	<2cm/hour

Protracted dilatation $\xrightarrow{\text{means}}$ Slow dilatation
 <1cm/hour for >4hour

Arrest disorder

- Arrest of dilation $\rightarrow$ No dilation for >2hours
- Arrest of descent $\rightarrow$ No descent for >1hour

Obstruction labour

00:02:25

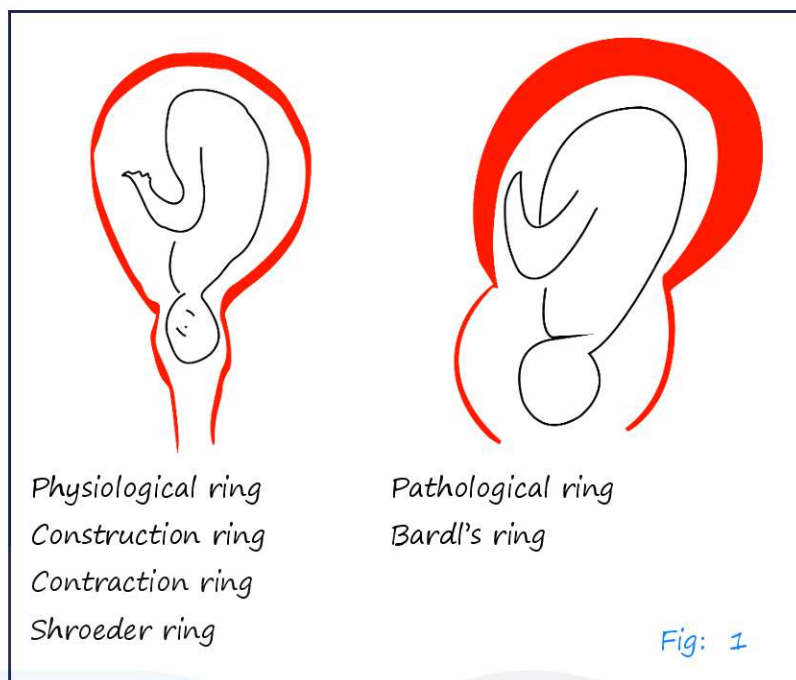
- In Spite of good uterine contractions, progressive descent of presenting part is arrested due to mechanical obstruction

Causes

CPD	Located twins
Contracted pelvis	Transverse lie
Prolapse	Brow
Fibroid	Hydrocephalus
Ovarian tumor	Ascites
Non grand horn of a bicornuate uterus	Occipito posterior position

Effects on mother and fetus

On mother	On fetus
Exhaustion	Asphyxia
Dehydration	Acidosis
Acidosis	Intra cranial infection
Sepsis/ PPH/ Shock	Injuries
Injury to genital tract	



	Features	Constriction ring	Retraction ring
P/A	Uterus	Firm/non tender	Tense and tender
	Fetal parts	Easily felt	Not felt
	Ring	Not felt	Felt
	Round ligament	Not felt	Felt
	FHA	+	Absent
P/V	Ring	Felt	Not felt
	Obstructed labour features	Absent	Present
End	Maternal/ fetal	Maternal exhaustion/ fetal distress are late	Early features
	Uterus	No chance of uterine rupture	Uterine rupture at lower segment
	Treatment principle	To relax the ring followed by delivery of baby To cut the ring during LSCS	To relieve the obstruction

Precipitate labour

- Total duration of labour is <3hours

SHOULDER DYSTOCIA CONTRACTED PELVIS AND CPD

Shoulder Dystocia:

00:00:30

Definition: Inability to deliver the shoulder within 1 min of delivery of head

Engaging diameter: Biacromial – 12 cm

Cause:

Macrosomia	prolonged labour
Diabetes	Multiparity
Obesity	Anencephaly
Post term	Fetal ascites

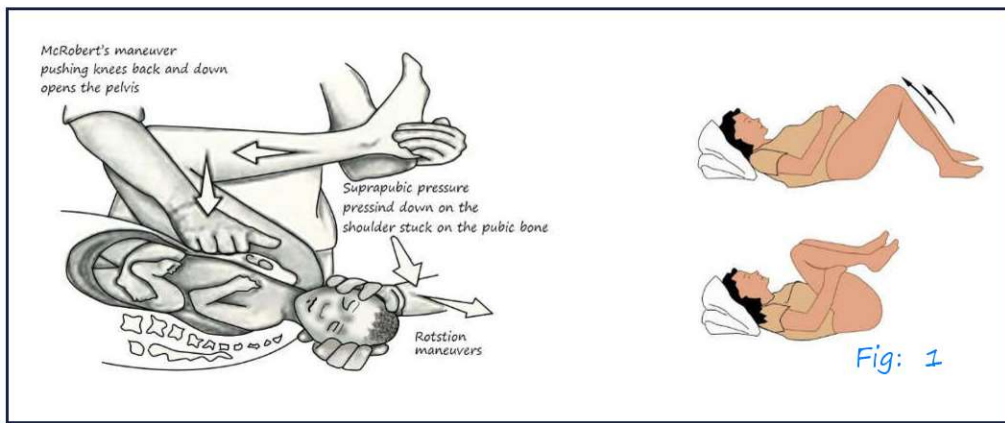
Turtle Neck Sign

- Head recedes back into the pelvis when trying to pull out

LOGIN FOR SUCCESS

Maneuvers

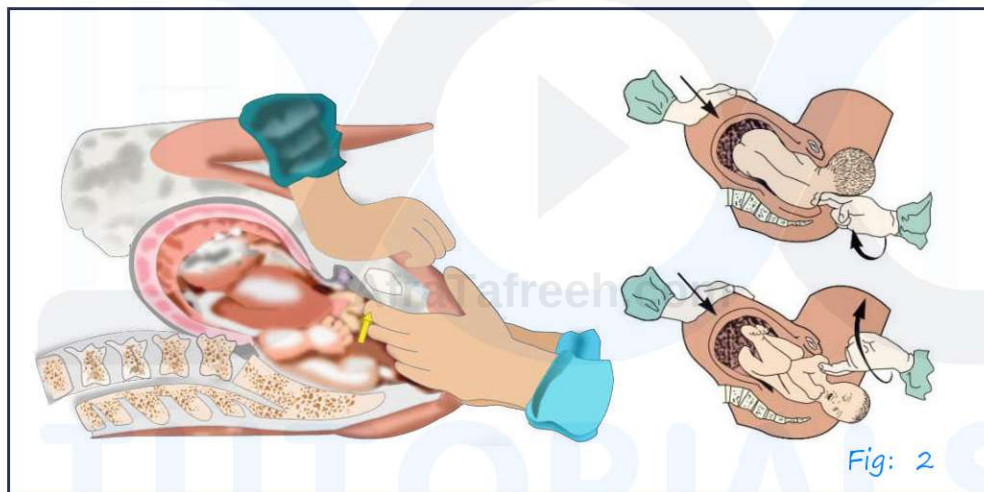
1. Mc – Roberts – Hyperflexion & abduction of maternal thighs



2. Rubin's I – suprapubic pressure

3. Rubin's II – Pressure on posterior shoulder P/V

4. Wood corkscrew – Rotate 180°



5. Gaskin – all four limbs posture /Dogs' posture

6. Zavanelli's – push the head back into pelvis & do

C-section

Complication:

Fetal

Fetal asphyxia
Erb's palsy
Klumpke's
Humerus fracture
Clavicle fracture
Sternomastoid – hematoma

Maternal

Injury to birth canal
Sacral
Iliac joint Dislocation

Q. Mc Robert maneuver → which nerve is commonly injured?

→ Lateral cutaneous nerve of thigh.

- Abdominal vaginal technique - Munro Muller method



TUTORIALS

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EPISIOTOMY

Episiotomy

- Definition: Surgically planned incision on the perineum and the posterior vaginal wall during the 2nd stage of labour is called Episiotomy.

Advantages

1. Clear and controlled incision – easy to repair.
2. Shortens the duration of 2nd stage
3. Reduces trauma to pelvic floor muscles, thus reducing the incidence of prolapse and incontinence.
4. Minimises intracranial injuries to the fetus, especially in preterm fetuses.

Indications

00:05:20

1. Shoulder dystocia
2. Breech
3. Fetal macrosomia
4. Operative vaginal delivery (forceps/vacuum)
5. Persistent occipito-posterior position
6. Short perineal length.
7. Suspected perineal injury.
8. Prolonged second stage of labour
9. Preterm baby
10. Fetal distress.

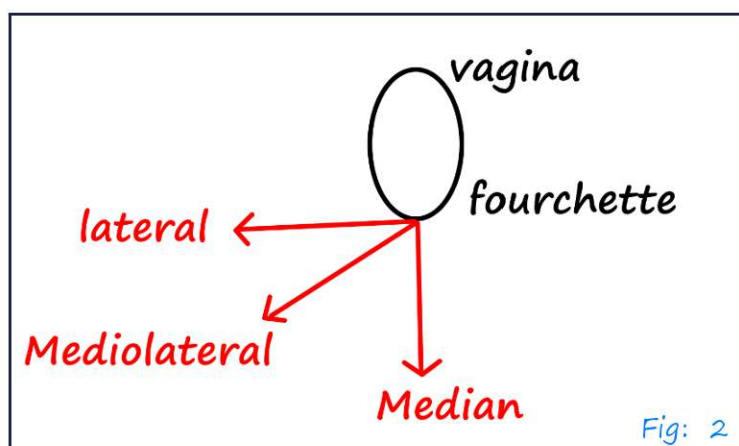
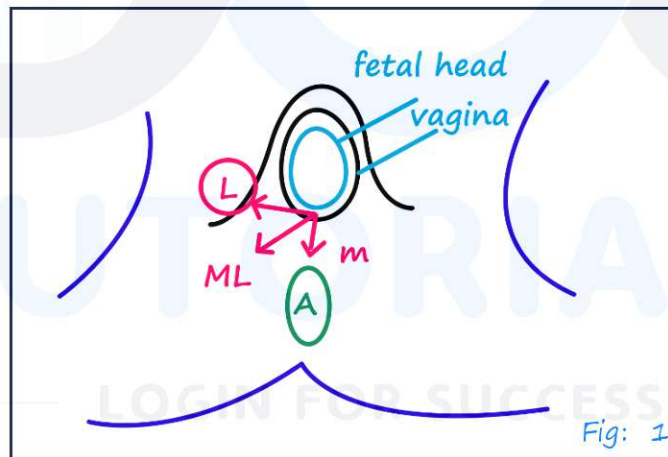
Timing:

- Ideal time → during crowning
- Crowning: When the head is visible to a diameter of 3-4cm^Q.
- Forceps – Episiotomy is given after the application of blades.

Types of Episiotomies

00:10:53

1. Median
2. Mediolateral
3. Lateral
4. J shaped

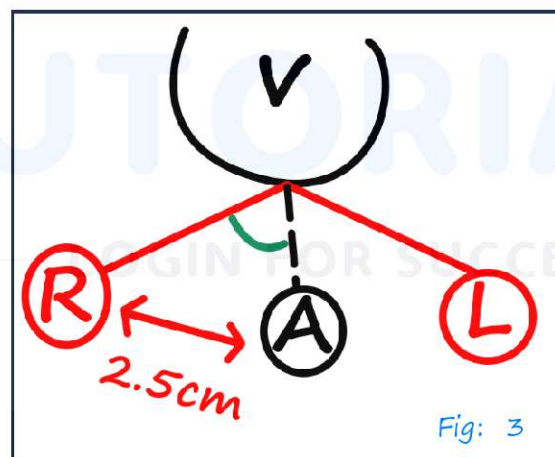


1. Median Episiotomy

- Starts from the centre of fourchette, extends posteriorly towards anus about 2.5cm in the midline.
- Incises the perineal body.
- Highest risk of sphincter injury.

2. Mediolateral

- Starts from the centre of fourchette.
- It can be on the right or left side
- Runs 2.5cm away from anus.
- Ideal cutting angle to prevent anal sphincter injury is 60°
- Angle is 45° off the midline for suturing.



3. Lateral

- Begins 1 cm away from the centre of fourchette.
- Angled towards the Ischial tuberosity
- Injures the Bartholin's duct

Structures cut in episiotomy

00:20:22

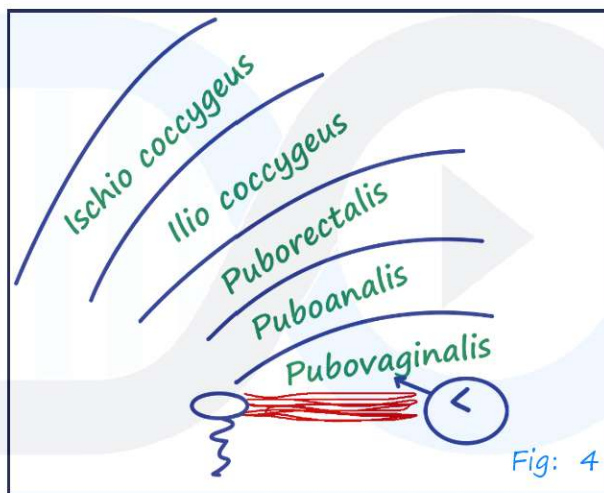
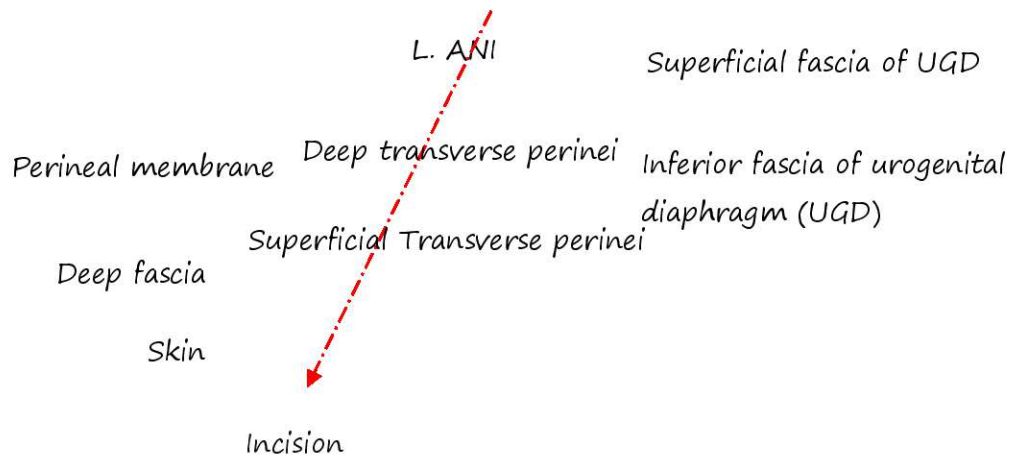


Fig: 4

- In mediolateral episiotomy which part of the elevator is cut?

→ Pubo vaginalis.

- In median Episiotomy → Perineal body is cut.

Pubo vaginalis fibres of levator ani

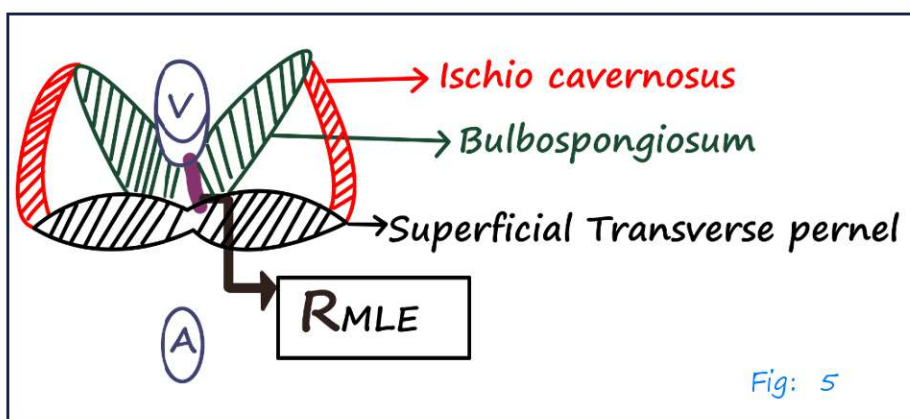


Fig: 5

Structures cut during episiotomy are

1. Bulbospongiosus
 2. Superficial transverse perinei
 3. Deep transverse perinei
 4. P. Vaginalis part of L. ani
- } 4 muscles

(Other structures)

5. Fascia covering these muscles
6. Transverse perineal branch of pudendal vessels and nerve
7. Subcutaneous tissue and skin.

Muscles of Perineal body

00:32:45

- Bulbospongiosus
 - Superficial transverse perinei
 - Deep transverse perinei
 - Pubo vaginalis part of levator ani
 - External anal sphincter
 - Sphincter urethra in males
- } Cut in episiotomy

Q. Which structures of the perineal body are not cut in Episiotomy?

Ans: External anal sphincter

Merits & Demerits

<i>Median</i>	<i>Mediolateral</i>
1. Less blood loss	More blood loss
2. Better healing	Apposition is not good poor healing.
3. Repair is easy	Repair is difficult
4. Less dyspareunia	More dyspareunia
5. Less postoperative discomfort	More discomfort
6. Increases risk of rectal or anal injury Injury to anal sphincter	Less risk of anal/rectal injury
7. Cannot be extended	Can be extended

Steps of Repair

00:38:55

- After expulsion of placenta.
 1. Vaginal mucosa/submucosa tissue.
 2. Perineal muscle
 3. Skin/subcutaneous tissue
- Suture material: Absorbable chronic catgut.
Episiotomy → 2nd degree perineal tear.

Complications

- Immediate complications

1. Injury to anal mucosa/rectum
2. Injury to sphincter.
3. Vulval hematoma
4. Wound infection
5. Wound dehiscence
6. Recto vaginal fistula
7. Necrotising fascitis.

- Remote complications

1. Dyspareunia
2. Scar endometriosis
3. Perineal tear in subsequent pregnancies.

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TUTORIALS

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CAESAREAN SECTION

Definition:

00:00:10

operative procedure whereby the fetuses are delivered through an incision on the abdominal wall and uterine wall after the period of viability.

Before the period of viability → hysterotomy

Primary C-section:

First caesarean section performed on a patient

Repeat C-section:

Caesarean section performed on subsequent pregnancies after a primary c – section.

Factors responsible for increasing caesarean section:

- Rising incidence of primary caesarean delivery
- Identification of at risk fetuses before term
- Identification of high risk pregnancy
- Wider use of repeat c section
- Rising rates of induction of labor and failure of induction
- Decline in operative vaginal delivery (Mid force caps, vacuum) and manipulative vaginal delivery
- Decline in vaginal breech delivery

- Increased number of women with age > 30 year and associated medical complications. Most are nulliparous
- Adoption of small family norm neither the obstetrician nor the patients are ready to accept any risk of abnormal labor
- Wider use of electronic fetal monitoring and increased diagnosis of fetal distress
- Fear of litigation in obstetric practice
- Caesarean delivery on maternal request

Elective Vs emergency C - section

00:07:52

Elective C – section

It is performed in a prefixed time

Advantages:

- Nil per oral (NPO)
- Good anesthesia
- Good facilities to resuscitate the neonate
- No pelvic floor dysfunction
- No risks related to labor

Emergency C – section

According to NICE Guidelines, four categories are there

- Category 1:

- There is immediate threat to the life of mother or fetus

Decision – delivery interval time should be within 30 mins

Example: – fetal distress

- late deceleration during labor
- atypical variable decelerations
- cord prolapse

- Category 2

- There is maternal or fetal compromise which is not immediately safe threatening

Decision – delivery interval time should be within 75 minutes

Example: cord presentation.

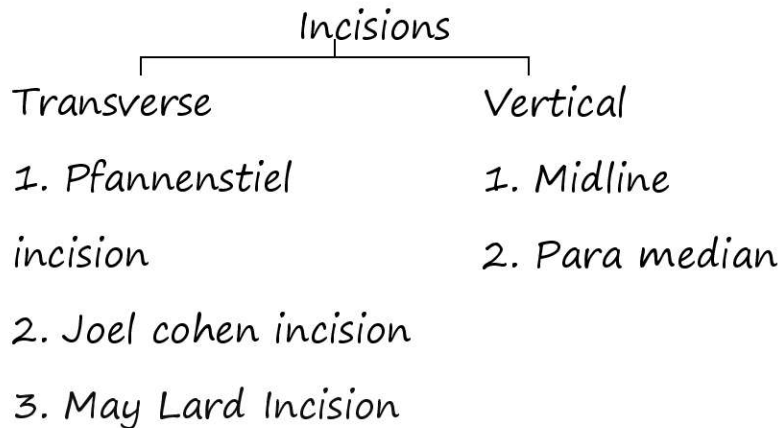
- Category 3

- No maternal/fetal compromise but needs early delivery

Example: severe preeclampsia

- Category 4

- Delivery is planned to suit the women, family members and the hospital staff.

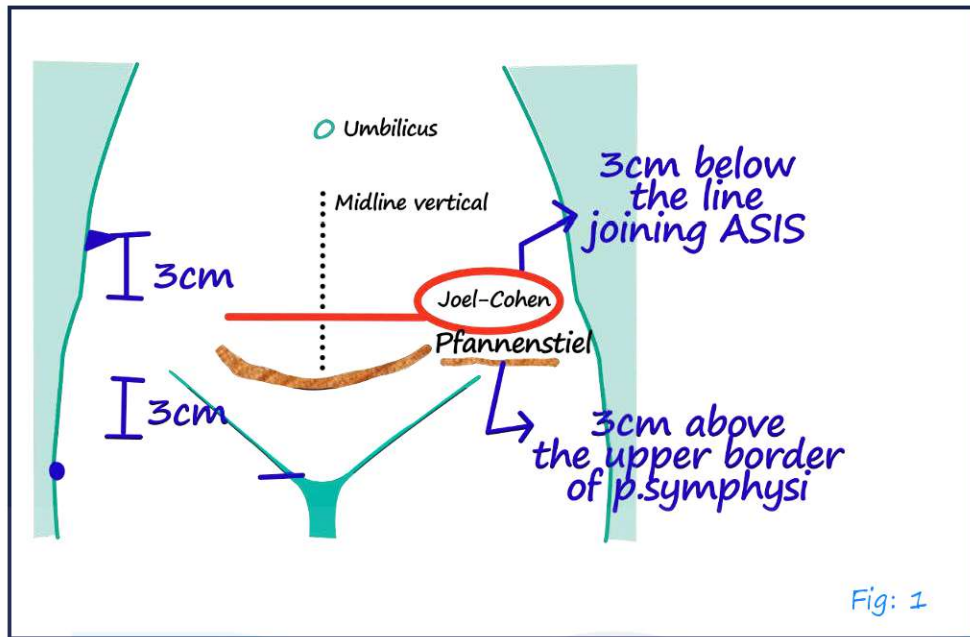


Differences between transverse and vertical incisions

	Transverse	Vertical
cosmetic	Good	Not good
Post operative pain	Less	More pain
Wound dehiscence	Less	More
Incisional hernia	Less	More
Skill required	More	Less
Access to upper abdomen	Less	good
Time taken for surgery	More	Less
Neurovascular injury	More	less

Transverse incisions:

- i. P. Pfannenstiel incision
- ii. Joel cohen incision
- iii. May lard incision



Differences between Pfannenstiel and Joel Cohen incision

P Pfannenstiel incisions	Joel Cohen incision
Low transverses Curvilinear/smiley incisions	low transverse <u>straight</u> incisions
3 cm above the upper border of pubic symphysis	3 cm below the line joining ASIS (anterior superior iliac spine)
Measure 12-15cm	Measures 10cm
More of strap dissection Rectus sheath cut with peritoneum Scissors	More of blunt dissection Rectus sheath is separable with figures Peritoneum cut with scissors.

Maylard incision:

Transect /cut rectum muscle Q

Ⓜ → Ⓜ

Maylard muscle

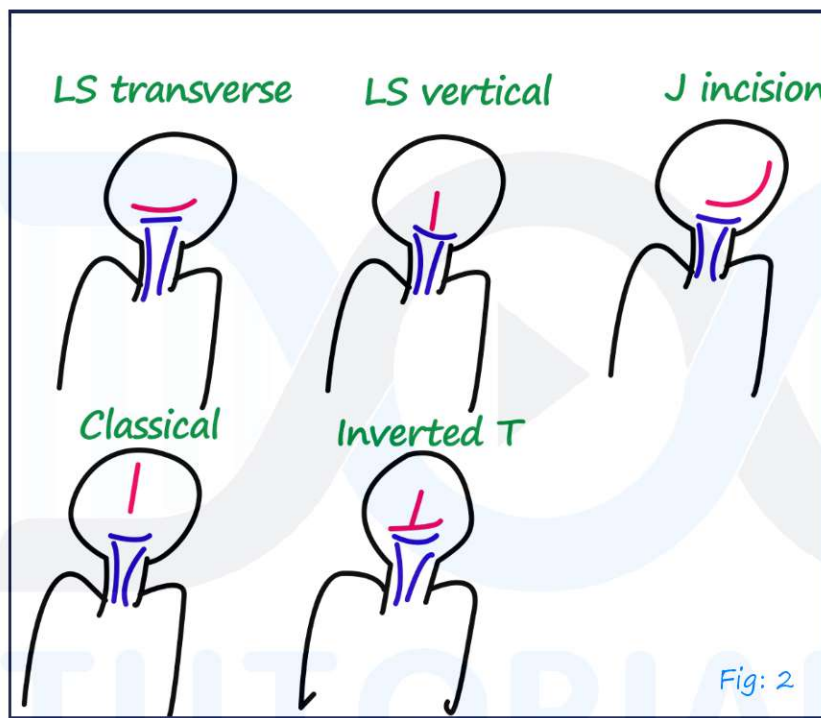
MCQs:

- i. Neurovascular structures injured in suupro pubic traverse incision are
 - Ilio-inguinal nerve
 - Ilio- hypogastric nerve
 - Superficial epigastric vessels
 - Inferior epinasties vessels
- ii. In which incision inferior epigastric vessel injured?
 - Pfannenstiel incision
- iii. What is misgav- ladach technique
 - Similar to Joel Cohen technique except for the peritoneum which is dissected without scissors here.
- iv. Most commonly used incision – pfannenstiel
- v. Incision used for emergency entry – midline vertical incision
- vi. Best incision for morbidly obese patient
 - Midline vertical incision
- vii. In which incision – rectus muscle is cut?
 - Maylard incision

Types of uterine incision

00:49:27

- i. Lower segment transverse incision □ most common.
- ii. Lower segment vertical incision
- iii. 'J' shaped incision
- iv. Classical incision
- v. Inverted 'T' incision



Indications for C – section

00:53:32

i. Maternal

- Prior caesarean delivery
- Abnormal placentation
Example: placenta previa and placenta accreta.
- Maternal request
Caesarean done on maternal request (CDMR)
- Prior classical hysterectomy
- Unknown uterine scar type

- Uterine incision dehiscence
- Prior full thickness myomectomy
- Genital tract obstructive mass
- Invasive cervical cancer
- Prior trachelectomy □ surgery to remove cervix
- Permanent cerclage
- Prior pelvic reconstructive surgery
- Prior significant perineal trauma
- Pelvic deformity
- HSV or HIV infection
- Cardiac or pulmonary disease
- Cerebral aneurysm or arteriovenous malformation
- Pathology requiring concurring intra abnormal surgery
Example: ovarian cyst
- Perimortem caesarean delivery

ii. Maternal – fetal indications:

- Cephalopelvic disproportion
- Failed operative vaginal delivery
- Placenta previa or placental abruption.

iii. Fetal indications:

- Non reassuring fetal status
Example: late/atypical variable deceleration and prolonged deceleration
- Malpresentation

- Macrosomia
- Congenital anomaly
- Abnormal umbilical cord doppler study
- Thrombocytopenia
- Prior neonatal birth trauma

Indications for classical C-section ^Q

1. Unapproachable lower segment (Densely adherent bladder)
2. Fibroid in the lower segment
3. Cervical cancer
4. Massive maternal obesity
5. Anterior placenta previa (Especially with p. accrete)
6. Transverse lie with ruptured membranes with and impacted shoulders
7. Entrapped fetal head or very small fetal in breech
8. Malpositioned twin and preterm twin
9. Conjoint twin

LOGIN FOR SUCCESS

Merits and demerits of LSCS over classical C –

Section

01:06:37

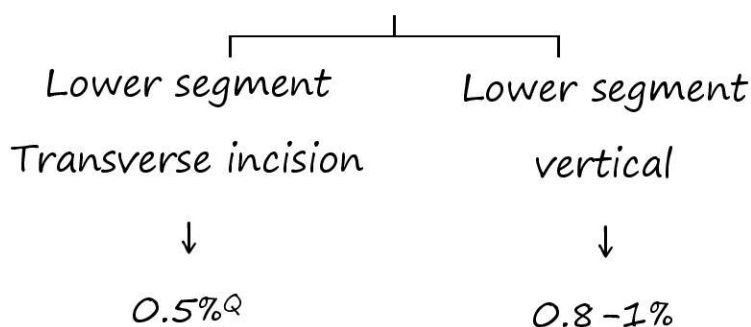
	Lower segment (Transverse)	Classical
Techniques	• Technically slight difficult • Blood loss is less	• Technically easy • Blood loss is more

	• The wall is thin & such as apposition is perfect • Perfect peritonization is possible • Technical difficulty in placenta previa or transverse lie	• The wall is thick & opposition of the margin is imperfect • Not possible • Comparatively safer in such circumstances
Post – Operative	• Hemorrhage and shock – less • Peritonitis is less even in infected uterus because of perfect peritonization and if occurs, localized to pelvis • Peritoneal adhesions and intestinal obstructions are less • Convalescence is better • Morbidity and mortality are much lower	• More • Chance of peritonitis is more in presence of uterine sepsis • More because of imperfect peritonization • Relatively delayed • Morbidity and mortality are high

Merits & Demerits of LSCS over Classical CS:

	<i>The scar is better healed because of:</i>	<i>The scar is weak because of:</i>
Wound healing	• Perfect muscle apposition due to thin margins • Minimal wound hematoma • The wound remains quiescent during healing process • Chance of gutter formation is unlikely	• Imperfect muscle apposition because of thick margins • More wound hematoma formation • The wound is in state of tension due to contraction and relaxation of the upper segment. As a result, the knots may slip or the sutures may become loose • Chance of gutter formation on the inner aspect is more
During future pregnancy	• Scar rupture is less (0.5 – 1.5%)	• More risk of scar rupture 4–9%

Incidence of scar dehiscence or rupture in subsequent pregnancy



Classical (4-9%)

Merits and demerits of LSCS over lower segment vertical incision

01:14:04

Parameters	Lower Segments Transverse	Lower Segments Vertical
Extension of incision	May occur to involve the uterine vessels	May occur to involve upper segment or downward the bladder
Bladder dissection	Minimal	More when extends inferiorly
Uterine closure	Easy	Difficult
Muscle apposition	Good	Often imperfect
Reperitonization	Complete	Often imperfect
Intraoperative bleeding	Less	More

Subsequent bleeding	Less	More
Risk of scar rupture	0.5 – 1.0%	0.8 – 1.1%

Intra operative and postoperative complications

01:18:15

Intra operative complications:

- i. Extension of uterine vessels
- ii. Injury to uterus and its vessels
- iii. Injury to bladder and ureter
- iv. GI Tract injury → bowel perforation
- v. Hemorrhage

Postoperative complications:

- i. Postpartum hemorrhage
- ii. Shock
- iii. Anesthetic complications
- iv. Infections
- v. Intestinal distractions
- vi. Deep vein thrombosis/thromboembolism
- vii. Wound complications
- viii. Ruptured uterus
- ix. Incisional hernia
- x. Chronic back pain/pelvic pain
- xi. Intentional obstruction due to adhesences

Recent INICET^Q → What is class 1 Robsons?

- Class 1 → nulliparous single cephalic, ≥ 37 weeks, spontaneous labor
- Class 2 → nulliparous single cephalic, ≥ 37 weeks, induced labor
(or) C-section done before labor
- Class 3 → multiparous single cephalic, ≥ 37 weeks, spontaneous labor
- Class 4 → multiparous single cephalic, ≥ 37 weeks, induced labor
(or) C-section done before labor
- Class 5 → multiparous single cephalic, ≥ 37 weeks, previous C-sections.
- Class 6 → All nulliparous breech
- Class 7 → All multiparous breech (including previous C - section)
- Class 8 → All multiple pregnancies (including previous C - section)
- Class 9 → All abnormal lie (including previous C - section)
- Class 10 → All single cephalic preterm pregnancies (including previous C - section)

POSTPARTUM HEMORRHAGE

Most common cause of maternal mortality

Definition

00:01:29

- Blood loss in
- Vaginal delivery – 500 ml
- Cesarean section – 1000 ml
- Cesarean hysterectomy – 1500 ml
- Fall in hematocrit – 10%

Types

00:02:26

- Primary PPH → Bleeding within 24 hours of delivery
- Secondary PPH → Bleeding after 24 hours but within 12 weeks

Causes

00:03:21

- Multipara
- Overdistension
- Malnutrition / anemia
- APH
- Prolonged labour
- Oxytocin
- Anesthesia
- Fibroid
- Uterine anomalies

- Tocolytics
- Precipitate labour –duration of labour is less than 3hrs
- Mismanaged 3rd stage
- Trauma / instrumental delivery

AMTSL

00:06:50

1. Inj. Oxytocin 10 IU IM within 1 minute of delivery of baby
2. Controlled cord traction → one hand to push the fundus above and other hand to pull the cord below



To prevent inversion

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4 T's

- Tone → Atonic PPH (Most common type) (Most common cause)



Most commonly leads to 1^o PPH

- Trauma → Instrumental /cs
- Tissue → Retained placental bits → 2^o PPH
- Thrombin → DIC

DIC in OBS

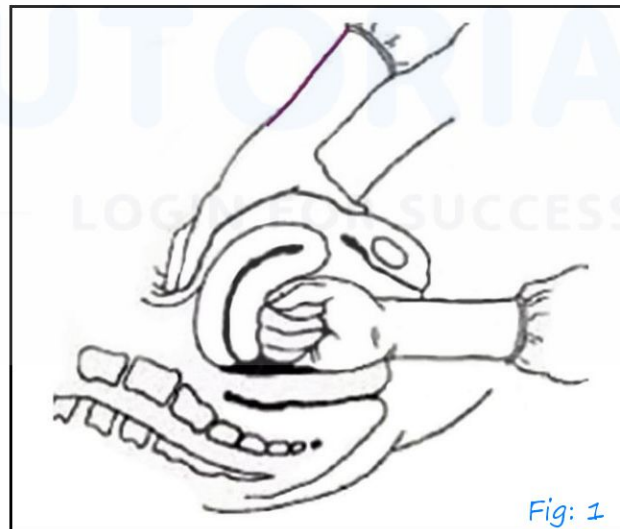
- IUD
- Pre-eclampsia
- Amniotic fluid embolism

- Sepsis
- Abruptio placenta

Management

00:14:56

- HELP
- 2 wide bore IV cannula
- Crystalloids (NS)
- O₂
- Vitals
- Uterine massage
- Bimanual compression
- Foleys tamponade
- Bakri balloon
- Bimanual compression



- Bakri balloon → Volume – 500 ml of saline injected

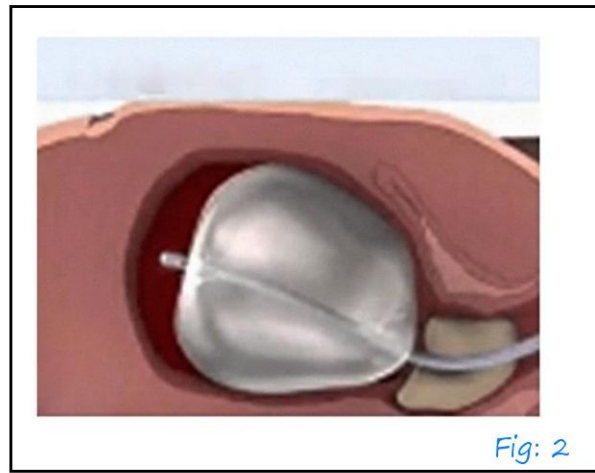


Fig: 2

- Management
 - Conservative/ preliminary
 - Medical
 - surgical

Medical

1. oxytocin
2. methyl ergometrine
3. PG $F_{2\alpha}$
4. PG E_2
5. PE E_1

Surgical

- Definitive → Hysterectomy

Conservative

1. Compression suture - B lynch, hayman
2. Ligation of arteries

Drug	Dose	Route	Frequency	S/E or C/I
Oxytocin	20U in 1L RL 200 mIU/mt	Infusion		Nausea, water intoxication, hypotension
Methylergometrine (methergine)	0.2 mg 200micg	IM/IV P/O	2-4 Hours	Hypertension, pre-eclampsia, women on HAART, heart disease
PGF _{2α} / carboprost	0.25 mg	IM	15-90 mts max 8 doses	Renal disease, liver disease, bronchial asthma, hypertension

PGE_2

- Gel form
- Dinoprostone
- 0.5 mg
- Used for cervical ripening in induction of labour

PGE_2	20mg	P/R		
PGE_1 (Misopros tol)	-R _x : 1000 micg - Prevention → 600micg	P/R		

Surgical management

- Compression sutures → B lynch → Named after Cristopher B lynch who used first



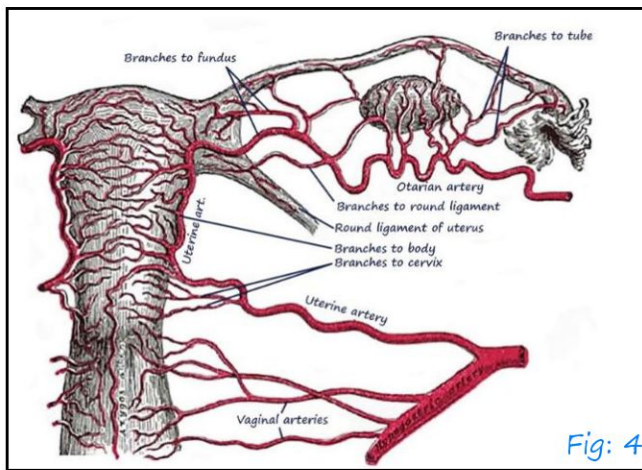
Ligation of pelvic vessels



Embolization



Hysterectomy



Ligate

00:33:22

1. Uterine artery
2. Ovarian artery
3. Anterior division of internal iliac artery
 - Definitive treatment of PPH – Hysterectomy

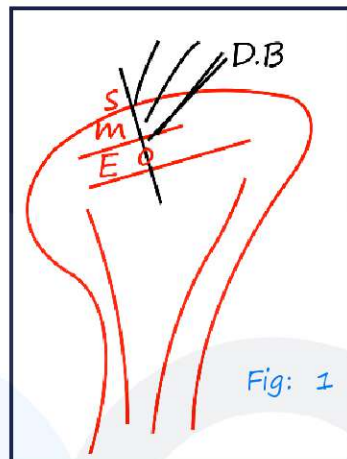
TUTORIALS

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PLACENTA ACCRETA AND RETAINED PLACENTA

On cut section through blastocyst

00:00:30



- Serosa
- Myometrium
- Decidua basalis/Endometrium
- Syncytio trophoblast → Nitabuch's membrane
- Cytotrophoblast
- Extra embryonic mesoderm

Nitabuch's Membrane

- Fibrinoid layer b/w syncytiotrophoblast & Decidua.
- Prevents syncytiotrophoblast (placental tissue) migrating into decidua.

Placenta accreta → placental tissue invades myometrium partially.

Placenta Increta → Placental tissue invades myometrium completely.

Placenta percreta → Placental tissue invades serosa ± bladder, rectum.

- If this Nitabuch's membrane layer gets destroyed due to prior surgeries, there is risk of placenta accreta.

Risk factors:

00:03:58

- Placenta previa
- Prior C-section → highest risk.
- Prior uterine surgeries
- Manual removal of placenta
- Synaecolysis (Adhesiolysis)
- Increases maternal age & parity
- Endometritis → (infection → scarring)

Investigation of choice:

- IOC: USG with Doppler.
- Gold standard Investigation: MRI
- Pathological confirmation: Decidua with absent Nitabuch's membrane

- If completed family → Hysterectomy
- If completed family & wants to retain uterus → Uterine artery embolization.
- If not completed family → Deliver the baby



Don't remove placenta, leave placenta insitu



Inj. Methotrexate



Lysis of placental tissue & shedding.



UTERINE INVERSION

- Mismanaged 3rd stage of labor is the important cause for inversion (Most common cause)

Risk factors

00:03:51

1. Fundal implantation of placenta
2. Uterine atony
3. Cord traction before placental separation
4. Placenta Accreta → Adherent placenta
5. Ehler Danlos Syndrome

Degrees

00:04:34

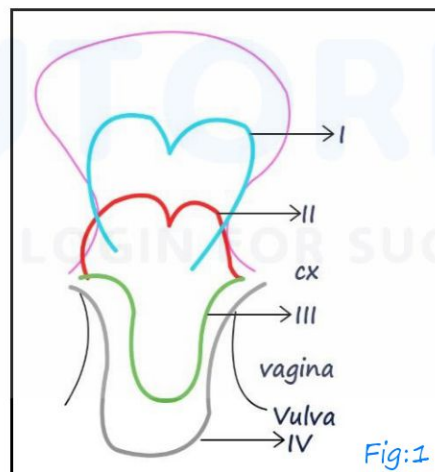


Fig:1

- 1st degree – dimpling
- 2nd degree – Fundus goes to the level of internal OS
- 3rd degree – Fundus inside vagina
- 4rd degree – Fundus outside introitus

- Call for help
 - ↓
- Check vitals
 - ↓
- Arrange for blood (>30% blood loss)
 - ↓
- Give general anesthesia
 - ↓
- Give tocolytics → Uterus relaxes
 - ↓
- Manual repositioning

Hydro static method ↓ Also called as 'O' Sullivans technique ↓ High speed water used to push the uterus back to its position	Abdominal method ↓ 1. Clamping and traction of round ligament ↓ Huntington's method 2. Sagittal posterior wall cut ↓ Haultin's method
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AMNIOTIC FLUID EMBOLISM

- Amniotic fluid embolism occurs when amniotic fluid, fetal tissues (vernix caseosa, lanugo hair) enter the material circulation
- Highly fatal
- Occur in intrapartum period/post-partum period
- Case fatality rate (CFR) – is as high as 11-43%
- Incidence – 1 in 40,000 to 1 in 50,000 deliveries

Pre disposing factors

00:02:36

• Rapid labour • Meconium stained liquor • Tears in uterine vessels • Eclampsia • Caesarean section • Forceps/vacuum delivery • Advanced maternal age	• Post term pregnancy • Induction of labour • Augmentation of labour • Abruptio placenta • Placenta Previa • Hydramnios
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Contraction- 35 to 40mmHg is where the blood flow completely ceases.

Diagnostic criteria

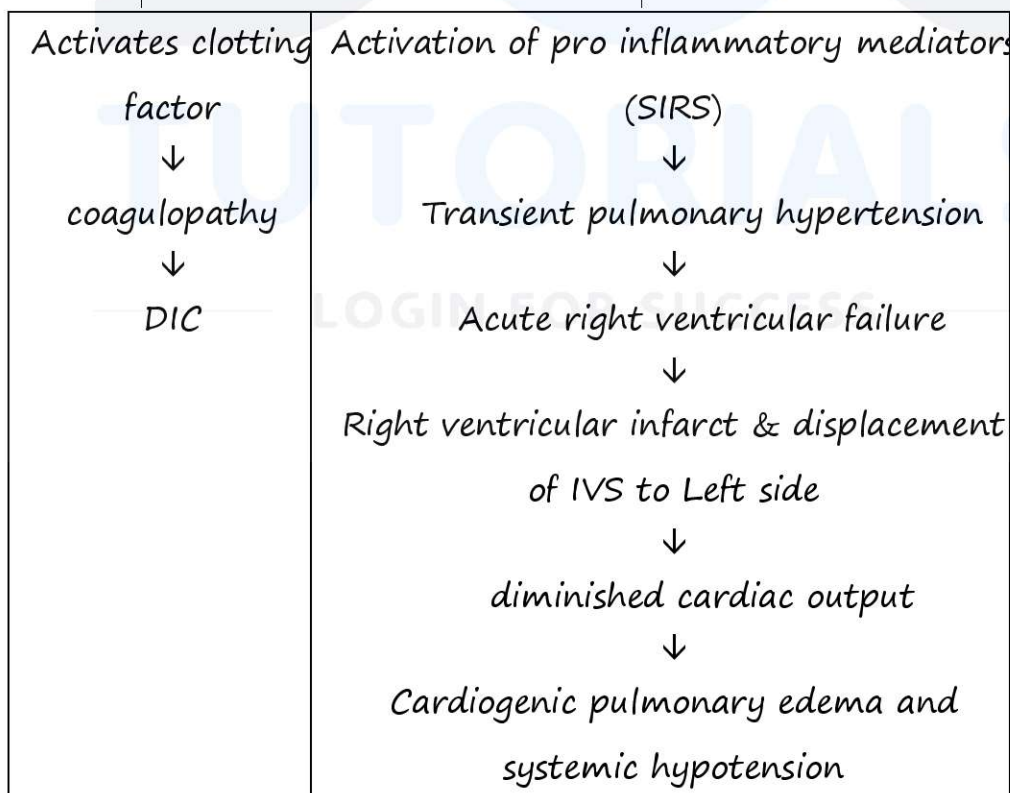
00:07:34

- i. Abrupt onset of cardio respiratory arrest
 - Hypotension/ Respiratory compromise
- ii. Overt DIC
- iii. Clinical onset during labour or within 30 min of placental delivery
- iv. No fever

Pathophysiology

00:10:00

Disruption of maternal foetal interface
material from fetal compartment enters maternal
circulation



Diagnosis- mainly clinical

- Autopsy/post-mortem evaluation (75% is positive)
- Ante mortem sampling of pulmonary artery to see Fetal tissue or vernix caseosa, lanugo or amniotic fluid (50% positive)

Treatment

00:17:20

- Supportive Measures
- For cardiac failure – Inotropes are given



3RD AND 4TH DEGREE PERINEAL TEAR

- Most common cause: Mismanaged 2nd stage of labour

Episiotomy causes 2nd degree perineal tear

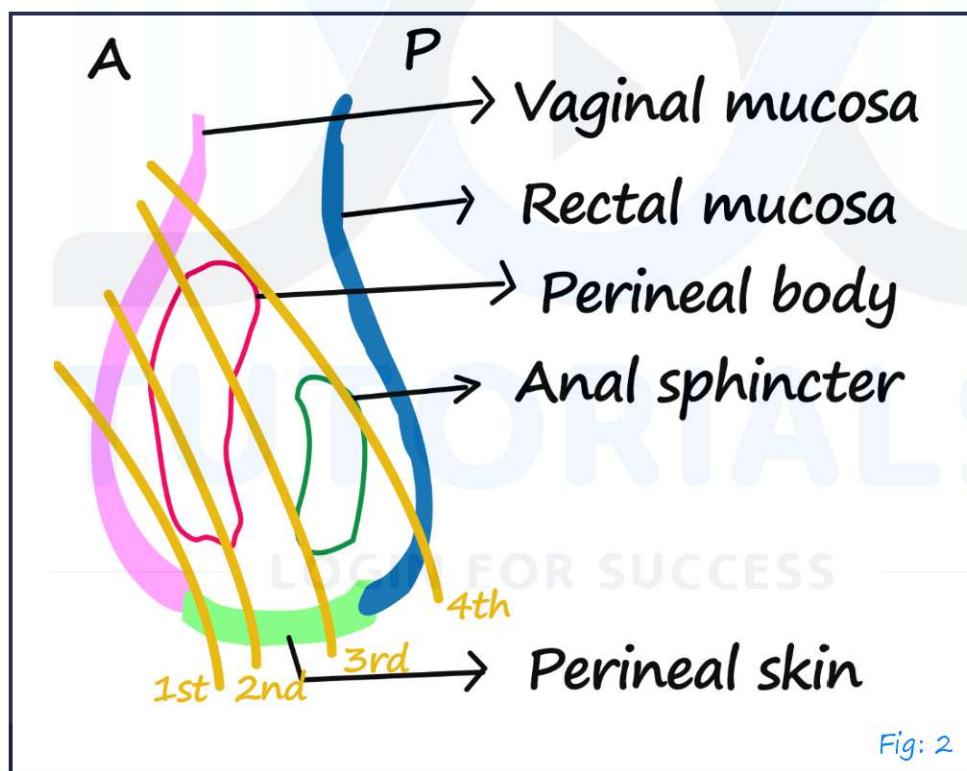
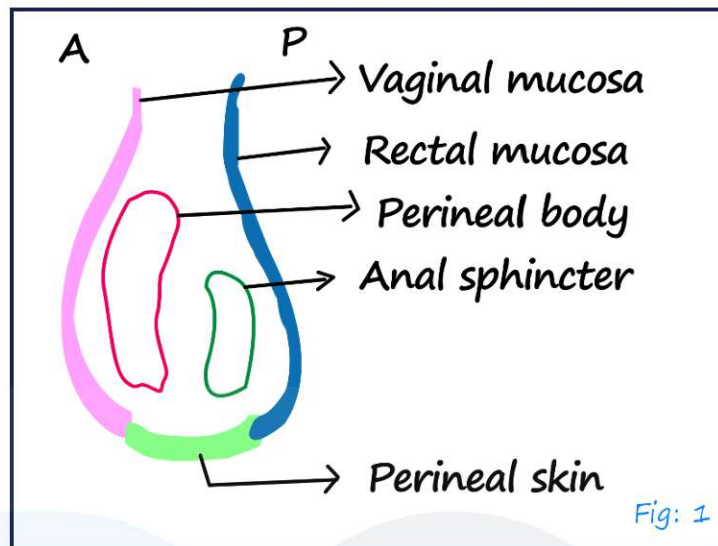
- It involves vaginal mucosa/ perineal skin and perineal muscles
- Episiotomy helps to avoid 3rd and 4th degree perineal tear
 - 3rd degree perineal tear → anal sphincter involved
 - 4th degree perineal tear → anal sphincter + mucosa involved

Predisposing factors AfraTafreeh.com

- Asian population
- Nulliparity
- Birth weight >4kg
- Shoulder dystocia
- Occipito-posterior position (face to pubis delivery)
- Prolonged 2nd stage of labour
- Instrumental delivery

Degree of perineal tear

00:04:25



Degree	Injury to
1 st degree	Perineal skin/ vaginal mucosa
2 nd degree	Perineal muscles
3 rd degree	Anal sphincter
3a	<50% of EAS torn
3b	>50% of EAS torn
3c	Both EAS + IAS are torn
4 th degree	Perineum involving anal sphincter and anorectal mucosa Complete perineal tear

EAS – External Anal Sphincter

IAS – Internal Anal sphincter

Rectal buttonhole tear

00:10:40

- Tear involving rectal mucosae with an intact anal sphincter complex
- It may lead to rectovaginal fistula.

OASIS

00:12:00

- Obstetric Anal Sphincter Injuries (OASIS)

Prevention

- Medio lateral episiotomy
- Perineal support during crowning (Ritgen manoeuvre)
- Warm compression
- Perineal massage during 2nd stage

Timing of repair

- Tear <24hrs old (within 24hrs of delivery) – Immediate repair
- Tear >24hrs old – Repair after 3 months

Technique and suture material

	Technique	Suture material
Anal mucosa	Continues (or) interrupted	3-0 vicryl
Internal anal sphincter (IAS)	Interrupted	3-0 PDS (or) 2-0 vicryl
External anal sphincter (EAS) i. Partial ii. Full thickness	- End to end repair - Overlapping repair	3-0 PDS (or) 2-0 vicryl

LOGIN FOR SUCCESS

Postoperative care and future deliveries 00:21:00

Post op care

- Nil per oral (NPO) – not required
- Laxatives/ antibiotics are recommended
- Physiotherapy
- 60-80% patient → Recover & asymptomatic in 12 months
- Recurrence rate in subsequent pregnancy → 5-7%

Future delivery

- OASIS → Not an indication for C-section in subsequent pregnancies
- OASIS → Not an indication for routine episiotomy in future pregnancies

Elective C-section in subsequent pregnancies

- For women who have suffered OASIS in previous pregnancy and who are
 - i. Symptomatic – (faecal incontinence)
 - ii. Abnormal endo anal ultrasound – external sphincter defect $>30^\circ$
 - iii. Abnormal manometry – squeeze pressure $<20\text{mmHg}$

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TUTORIALS

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DEFINITION AND TYPES OF ABORTIONS

Definition

00:00:24

- Spontaneous or induced termination of pregnancy before period of viability

Period of Viability

00:01:02

- ACOG – 20wks/<500g
- RCOG – 24wks/<500g
- WHO – 20wks/<500g?

Incidence → 11-22%

00:02:32

- Recurrent pregnancy loss (RPL):
 - 2 or more consecutive failed pregnancy confirmed by Ultrasound/HPE
 - Incidence of RPL
 - After 2 abortions – 1%
 - After 3 abortions – 30%
 - After 5 abortions – 50%

Primary RPL

- No live born babies

Secondary RPL

- 1 or 2 Live babies present

Types

00:05:25

1. Based on Trimester

- 1st trimester – 80%
- 2nd trimester – 20%

2. Based on Frequency

- Sporadic
- Recurrent

3. Based on Clinical features

- i. Threatened
- ii. Missed
- iii. Incomplete
- iv. Inevitable
- v. Complete

Abortion	Clinical feature	Size of uterus	Internal OS	USG/Treatment
Threatened	Spotting	Equal	Closed	Live fetus progesterone
Inevitable	Bleeding	Equal	Open	Dead fetus
Incomplete	Bleeding	Small	Open	Retained Products
Complete	Expulsion No bleeding	Small	Closed	Empty Uterus
Missed	Asymptomatic	Small/equal	Closed	Dead fetus

Criteria to diagnose abortion (or) blighted ovum

- G sac → Measuring ≥ 25 mm with no yolk sac
- CRL (Crown Rump Length) → ≥ 7 mm with no cardiac activity

Adverse outcomes of threatened abortion

- Placenta Previa
- Abruptio
- Manual removal of Placenta
- Preterm delivery/PROM
- IUGR
- IUD (Intra Uterine Death)
- Increase C. section

Causes of Abortions

00:16:02

Fetal cause

- i. Chromosomal abnormality
 - 50% of 1st trimester abortions are chromosomal abnormality → 95% Maternal & 5% Paternal
 - Trisomy is the most common abnormality causing abortion
 - Trisomy – 13, 16, 18, 21, 22
↓
 - MC – Among trisomy abortions → Trisomy 16 > 22
 - Monosomy → 45X (Turner's syndrome) → **Single M/C aneuploidy causing abortion**

Turner's Contribute – 9-13% of aneuploidies causing abortion

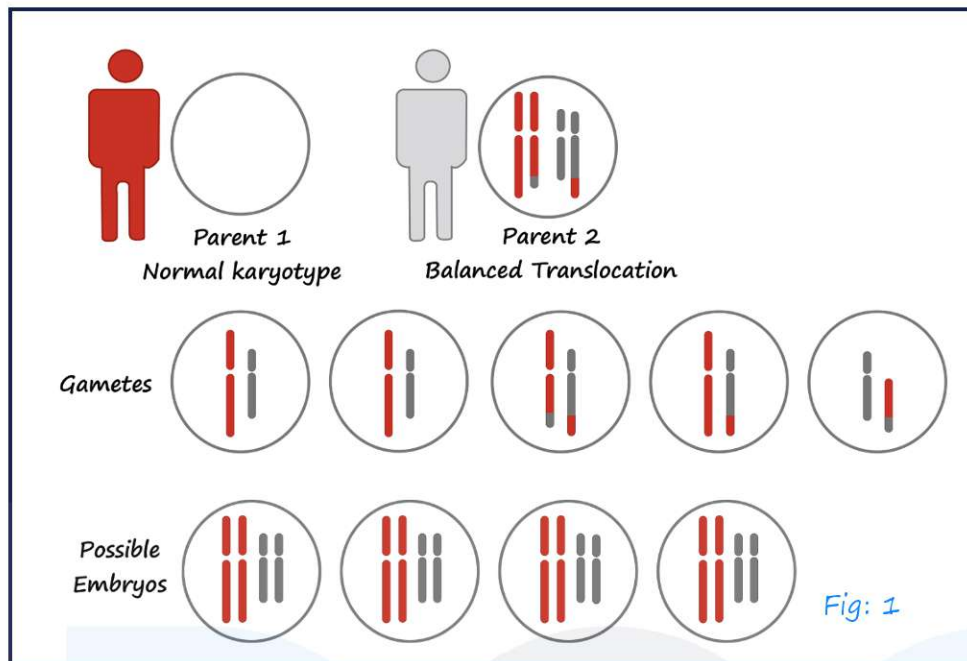
ii. Multiple pregnancy

Parental causes

00:20:48

- Chromosomal abnormality
- Anatomical factors
- Immunological factors
- Endocrine factors
- Chronic disease of the mother
- Thrombophilias
- Infection
- Addictive habits
- Environmental factors
- Others → Increased paternal age & Oophorectomy
 - Most common cause of Miscarriage → Chromosomal abnormalities (Aneuploidy)
 - Most common cause of 1st trimester miscarriage → Chromosomal abnormality (Aneuploidy)
 - Most common cause of 2nd trimester miscarriage → Cervical incompetence
 - Most common cause of recurrent miscarriage → Idiopathic > Cervical incompetence
 - Most common aneuploidy leading to abortion → Trisomy > Turners > Trisomy 16

1. Chromosomal Abnormality → Translocation involving either parent (Balanced translocation 2-4% of RPL)



2. Anatomical factors – Cervical incompetence and uterine anomalies

3. Immunological factors → APLA/SLE

4. Endocrine factors

- Diabetes mellitus
- Hypothyroidism, Hyperthyroidism
- Hyperprolactinemia
- PCOS

5. Chronic disease of mother

- TB
- Leukemia
- Congestive cardiac failure
- Renal failure

6. Thrombophilia

- Protein C/S deficiency, Factor V Leiden mutation

7. Thrombophilia

- Protein C/S deficiency, Factor V Leiden mutation

Septic abortion

00:33:59

- A temperature 100.4°C for $>24\text{hrs}$ following miscarriage

Grades

- Grade I $\rightarrow$ Infection limited to uterus
- Grade II $\rightarrow$ Infection to other parts of pelvis
- Grade III $\rightarrow$ Extra pelvic infection
- Grade IV $\rightarrow$ Shock

Investigation for Recurrent pregnancy loss (RPL)

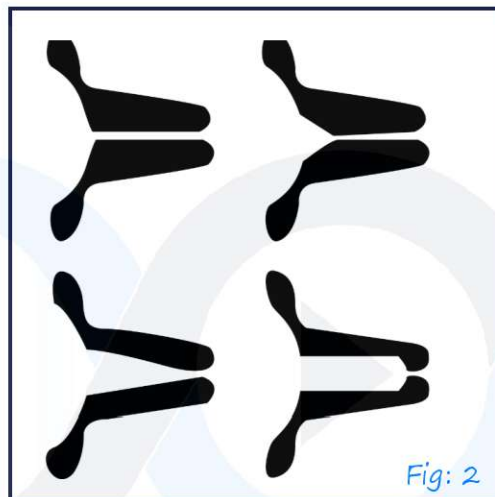
00:3815

- FBS, PPBS, HBA1C - DM
 - VDRL - For Syphilis
 - Thyroid Function Test
 - Prolactin
 - Blood grouping and typing
 - APLA screening
 - USG and Day₂ serum LH for PCOS
 - Hysterosalpingogram
 - Hysteroscopy
 - Diagnostic laparoscopy
 - Endocervical swab $\rightarrow$ For cervical infection
 - Karyotyping of both partners
- } For Uterine anomalies

Cervical incompetence

00:37:17

- Most common cause of 2nd trimester Miscarriage
- Definition
- Cervix measuring $< 2.5\text{cm}$
- Cervical OS open $> 1\text{cm}$
- Ideal time to measure cervical length – 24wks



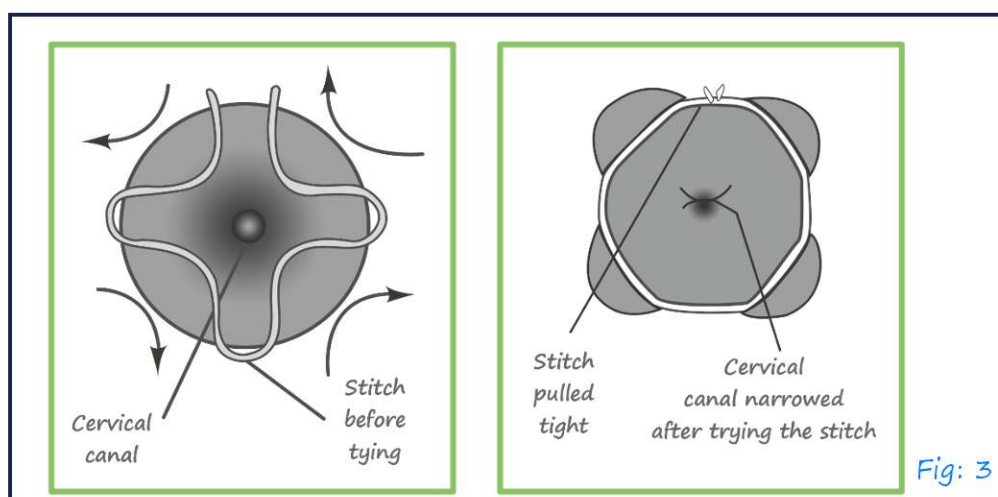
- Shape $\rightarrow$ "T" $\rightarrow$ "Y" $\rightarrow$ "V" $\rightarrow$ "U"
- "u"-severe form of incompetence

En-cerclage

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00:46:31

- MC Donald encerlage /Purse string suture



- Easy to apply
- M/C performed
- Shirodkar suture
- Advantage – Suture at internal OS bury thread under vaginal mucosa → (NO irritation)
- More effective

Ideal time to apply cerclage → 14wks

or

at least 2wks prior to earlier abortion

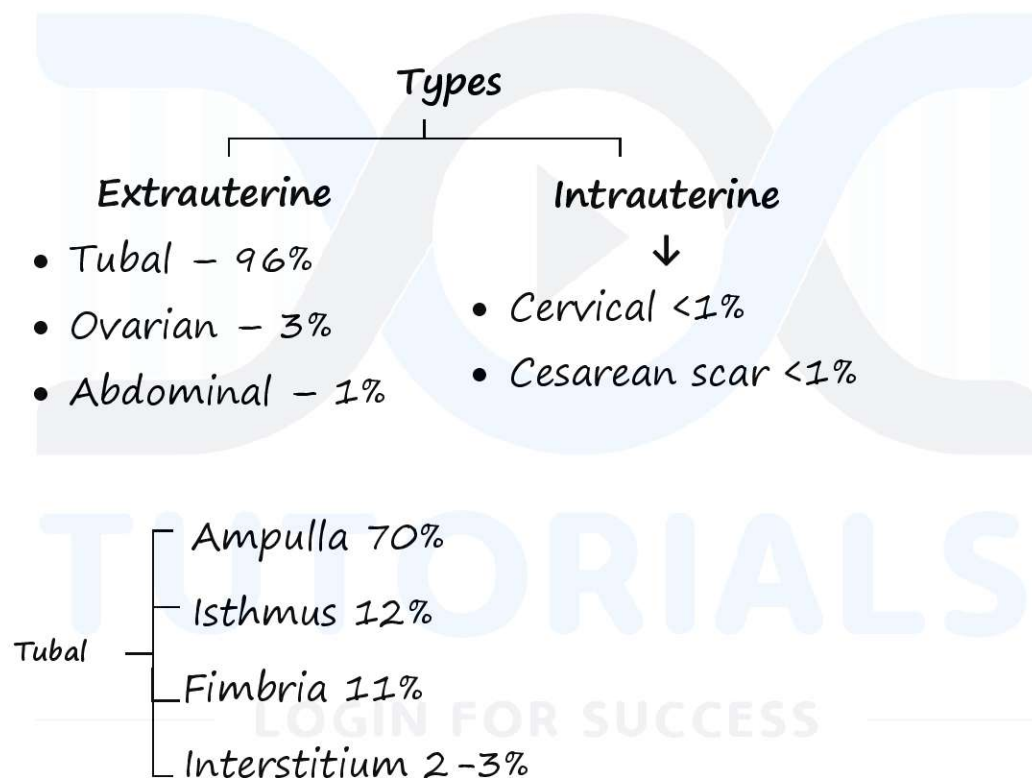
- When to remove → Completed 37wks
- Indications of cerclage
 - History alone → Optional
 - History + short cervix → Encerclage should be applied
 - Only short cervix → No use
 - Rescue cerclage → Yes
 - Twins → No use
 - Material Used → Prolene / Mersilene Tape

ECTOPIC PREGNANCY PART - I

- Common cause of maternal mortality

Definition:

- Implantation of the fertilized ovum occurring outside the normal uterine cavity
- 1 in 150 to 1 in 300 pregnancies



Risk Factors

00:05:58

- Past H/O ectopic pregnancy
- Prior tubal surgery → (4-fold ↑)
- Smoking (4-fold ↑)
- PID

- Q. M/C organism responsible for ectopic →
Chlamydia

v. ≥ 3 spontaneous abortion

vi. Age > 40

vii. MTP/IVF/Ovulation induction/Progesterone only pills

- Progesterone
- Smooth muscle relaxant
- Tubal motility
- Ectopic pregnancy

Pathophysiology

00:11:36

- Interstitial cells of kajal



Pacemaker cells of fallopian tube



(From where the contraction begins)

If get damaged



Motility of fallopian tube ↓

- Cannabinoid receptor



Smoking



Decreases tubal motility → Ectopic

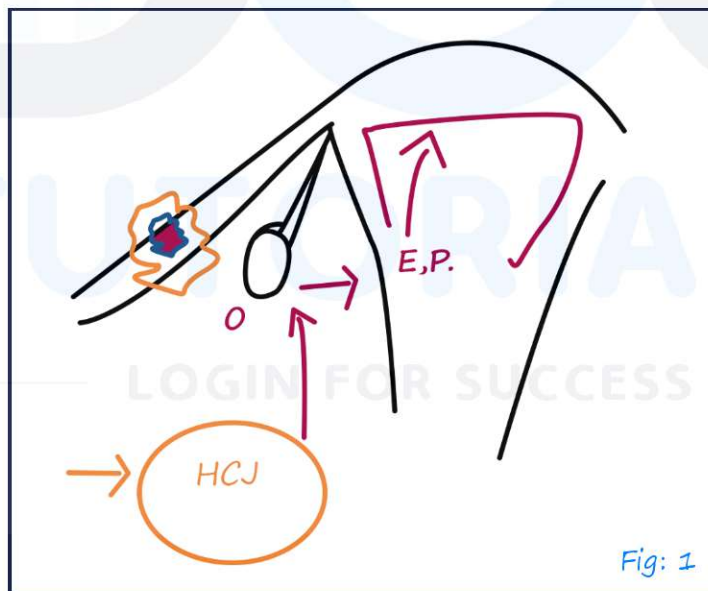
Clinical features

00:13:20

- i. Amenorrhea → M/C symptom
 - ii. Pain abdomen
 - iii. Bleeding (Minimal bleeding)
- } Triad of ectopic pregnancy

Diagnosis

- Syncytio trophoblast → Secrete – HCG
- ↓
- Stimulates Corpus luteum in ovary
- ↓
- Ovary secrete estrogen, progesterone
- ↓
- Endometrium – becomes thick



- In ectopic
 - HCG → More than non-pregnant/non-viable pregnancy but Less than Normal Intra uterine Pregnancy

IOC → USG



- True Gestational Sac → Evidence of Normal Intra uterine pregnancy
- Pseudo gestational Sac → evidence of Ectopic pregnancy

True Sac	Pseudo Sac
Double layered	Single layered
Regular	Irregular
Eccentric	Central
Yolk sac present	Absent
Increases by 1 mm/day	Does not increase by 1mm/day

Q. When G. Sac (+)/YS (-)/Embryo

00:26:55

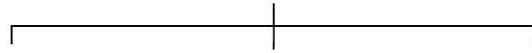
Inconclusive on USG



Gestational sac ⊕ / yolk sac/embryo ⊖



HCG (2 values – in 48 hrs)



Increases
& doubles



Normal
IUP

Increases but no
doubling



Ectopic
(growing/unruptured)

Decreases



Failing
IUP
Failing
Ectopic

S. Progesterone

- In normal Pregnancy → >10ng/ml

Culdocentesis

Pouch of Douglas / cul de – sac



Aspirate

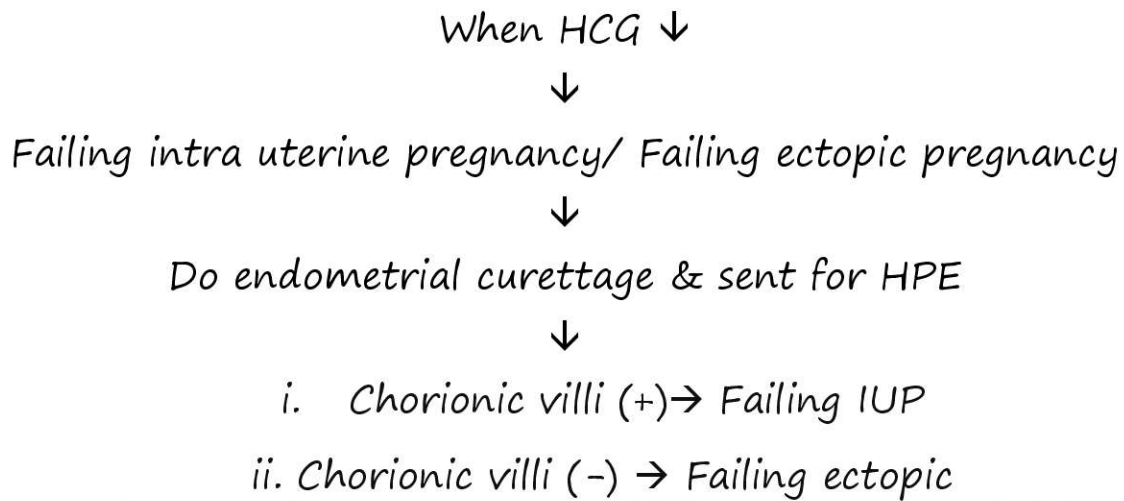


Bloody tap (Does not clot)



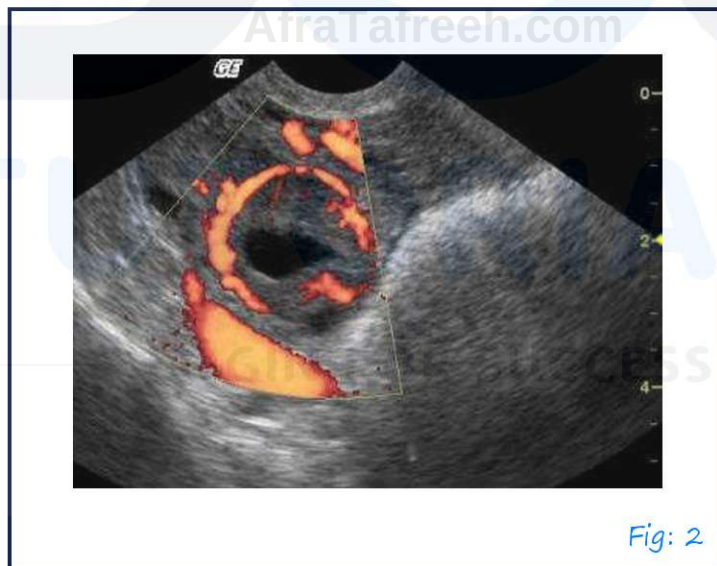
Probably from ruptured ectopic pregnancy

Endometrial curettage:



Laparoscopy

- Q. Gold standard investigation
- 100% confirmatory & Therapeutic



ECTOPIC PREGNANCY PART - II

Definition:

- Implantation of fertilized ova outside the normal endometrial cavity
- Incidence
 - 1 in 150 to 1 in 300 pregnancies
- Types
 - Tubal (96%)
 - Ampulla (70%)
 - Isthmus (12%)
 - Fimbria (11%)
 - Interstitium (2-3%)
 - Ovarian (3%)
 - Abdominal (1%)
 - Cervical (1%)
 - Cesarean scar (<1%)

Risk Factors

00:02:14

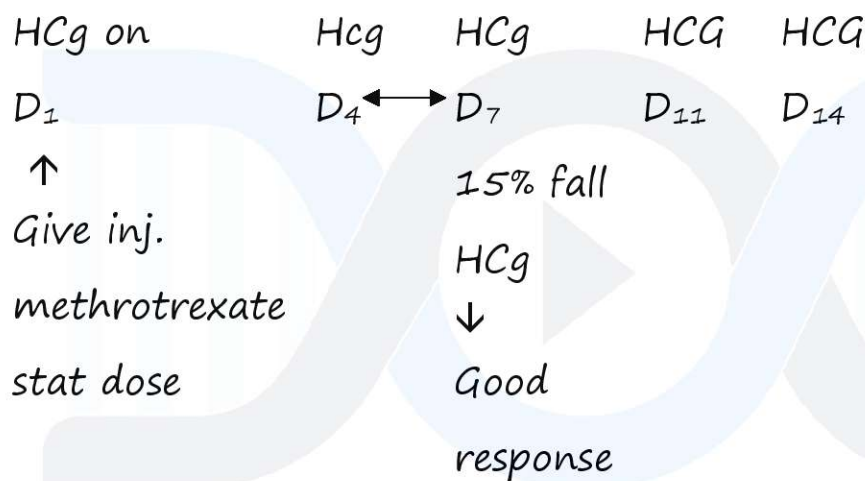
- Prior ectopic pregnancy (10-16 fold increases risk)
- Prior tubal surgery (4 fold increases risk)
- Smoking (cannabinoid receptor) (4 fold increase risk)
- PID → Chlamydia
- ≥ 3 sponaneous abortions
- Age > 40
- MTP/IVF/POP → progesterone inhibits tubal mobility

- Clinical Features
 - Triad of ectopic
 - Amenorrhea (M.c complaint)
 - Pain (Most consistent symptom)
 - Bleeding
- Diagnosis
 - TVS (IOC) whenever women comes with amenorrhea with – urine pregnancy test positive.
 - Adenexal G.sac (ectopic)
 - Inconclusive (s.βHCG) → Repeat after 4hours
 - If doubling + ~ intra uterine pregnancy
 - If doubling - ~ Ectopic or failing pregnancy

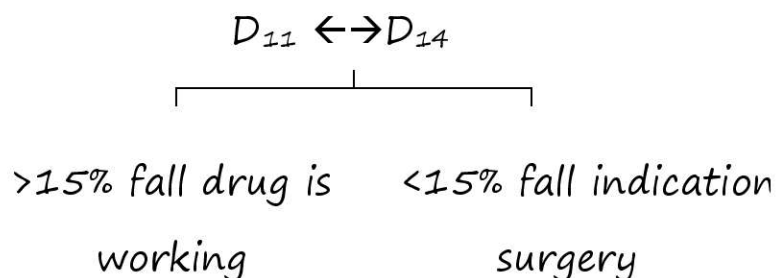
Progesterone:

- >5mg/ml – viable pregnancy
 - <5mg/ml – Non-viable pregnancy
 - Culdocentesis
 - Endometrial curettage
- Usual timing of rupture
- Isthmus → Earliest to rupture 8 wks
 - Ampulla → 12 weeks
 - Interstitium → 16 weeks – bleeding ++ Most dangerous type
 - Management
 - Expectant – falling βHCG with initial HCG < 100u
 - Medical management prerequisites

- i. Sac size $< 3.5\text{cm}$
- ii. HCG < 5000
- iii. Inruptured/stable mother
- iv. Tubal ectopic
- v. No cardiac activity
- Doc: methotrexate 1mg/kg around 50mg
- Measure



$>15\%$ fall — $<15\%$ fall $\rightarrow$ Repeat dose on
 satisfactory unsatisfactory D_7 methotrexate.
 on D_{11} , D_{14}
 $\rightarrow$ check HCG



Surgical Management:

- Laparoscopy (minimally invasive)
- Family completed → salpingectomy

Family not completed



Opposite tube



Cervical Pregnancy/Rubin's Criteria:

4 components

- Closed internal os and open external os
- Placenta below the entrance of uterine artery
- Cervical glands near placental attachment

Ovarian Pregnancy/Spiegelberg Criteria:

- Intact ovarian ligament
- Intact tube
- Ectopic occupying ovary with ovarian tissue in the placenta

Abdominal Pregnancy / Studio ford criteria:

- Intact uterus (No fistula)
- Intact tubes
- Intact ovaries
- Ectopic should occupy the peritoneal surface

Heterotrophic Pregnancy:

- Simultaneous occurrence of intrauterine & ectopic pregnancy

Management:

- Kcl is directly injected into tubes
SAM – (surgically assisted medical management)



<i>Placenta Previa</i>	<i>Abruptio placenta</i>
<i>Painless/causeless</i>	<i>Painful/hypertension/trauma</i>
<i>Always revealed</i>	<i>Concealed/revealed/mixed</i>
<i>Bright red</i>	<i>Dark</i>
<i>Anemia proportionates</i>	<i>Out of proportion in Concealed/mixed</i>
<i>No hypertension/PE</i>	<i>Hypertension/PE</i>
<i>Corresponding to GA</i>	<i>Enlarged in concealed</i>
<i>Soft and relaxed</i>	<i>Tense & tender</i>
<i>Malpresentation present</i>	<i>Absent</i>
<i>FHs preset</i>	<i>Absent</i>
<i>DIC absent</i>	<i>Present</i>
<i>Fetal parts easily palpated</i>	<i>Difficult to palpate</i>

TUTORIALS

LOGIN FOR SUCCESS

SICKLE CELL DISEASE IN PREGNANCY

Maternal and fetal complication

00:00:22

1. Preterm labour
2. Infection
3. IUGR
4. Thromboembolic event
5. Abortion
6. Post-partum Hemorrhage
7. Acute painful crisis
8. Pre-eclampsia
9. Frequent Hospitalization
10. Pulmonary hypertension
11. Increase rate of c-section

Maternal changes aggravating sickle cell disease

00:03:20

1. Dehydration (due to nausea/vomiting)
2. Anemia
3. Risk of infection

Pre pregnant Assessment:

1. Pulmonary hypertension
2. Blood pressure /proteinuria
3. Retinal screening
4. Iron overload

5.Red cell Alloimmunization

Iron overload: Aggressive Iron chelation should be done before conception

Pre pregnancy medication:

- 1.Penicillin prophylaxis (Hypersplenism)
- 2.Erythromycin – penicillin allergy
- 3.Vaccination – H. Influenza
 - Meningococcus
 - Pneumococcus
 - Hepatitis B
 - Swine flue
- 4.Folic acid 5 mg
- 5.Hydroxy urea/carbamide – stop at least 3 months before conception
- 6.ACE inhibitors /ARBs

Antenatal hemoglobinopathy screening

Sickle cell disease during pregnancy



Screen the partner for cancer state



Cancer +



Chronic villous sampling 10 weeks

Or

Amniocentesis 2nd trimester (15weeks)



Affected baby



Termination

Antenatal Medication

Penicillin

Folic acid

1. Iron supplementation only if deficient
2. Low dose aspirin
3. Prophylactic LMWH – hospitalized
4. Compression stocking

Blood Transfusion

Blood Transfusion → No rule for routine prophylactic transfusion

Twin pregnancy → prophylactic transfusion can be considered

Acute anemia → Top up transfusion

Comorbidities → Top up transfusion / exchange transfusion

Acute stroke

Or

Acute chest syndrome → exchange transfusion

Women who are on transfusion regimen before pregnancy → continued

- Mild pain → NSAID/ paracetamol – Avoid 1st & 3rd trimester
- Moderate Pain → weak opioid (Dihydrocodeine)
- Severe pain → strong opioid (Eg: Morphine)
- Pethidine → C/I on women with Sickle cell disease d/t Risk of seizures
- Most common complication/ Most common cause for hospitalization → Acute painful crisis
- 2nd MC complication for Hospitalization → Acute chest syndrome

Mode of delivery:

- Induction of labour → at 8 weeks
- Not a C/I for vaginal delivery /VBAC
- Labour → increase sickle cell crisis
- Anesthesia of choice → Regional anesthesia (c-section)

Post-partum:

1. LMWH prophylactic
- vaginal delivery → 7 days
 - LSCS → 6 weeks

2. Antithrombotic stockings

3. NSAID

Contraception

- *DOC: Progesterone (decrease sickling)*
- *Combined oral contraceptives: (E+P)*
2nd line agent.



IRON DEFICIENCY ANEMIA

Anemia in pregnancy:

00:00:34

- WHO Definition: Anemia is defined as Hemoglobin (Hb) ≤ 11 gm% (or) hematocrit of $< 33\%$
- CDC Definition:
 - 1st ,3rd Trimester $\rightarrow 11$ gm %
 - 2nd Trimester $\rightarrow 10.5$ gm%

Causes of Iron deficiency Anemia:

00:02:54

Inadequate intake	Diet low in Iron /Diet with low bioavailability
Iron loss	Hookworm infestation malaria
Low Iron stores	Short interpregnancy interval
Defective absorption	Vit A, Vit C, deficiency Phosphate, phytate
Menstrual disturbances	Heavy bleed

Hemoglobin changes pregnancy:

00:06:03

- Blood volume increased by 40-45%
- Begins to increase in the 1st Trimester
- Rate of increase is highest in 2nd Trimester
- Maximum a 32 -34 weeks

Blood volume	- increase 40-45 %
Plasma volume	- 40-50 %
Red cell mass	- 20%
Hematocrit	- decrease
Hb concentration	- decrease

- Hemodilution in pregnancy → physiologic anemia
- Physiological anemia – more in 2nd Trimester (Due to difference in plasma volume increase & Red cell mass increases in more)

Iron metabolism in pregnancy

- Daily Iron requirement → 1st, 2nd Trimester – 4-6 mg /day
→ 3rd Trimester – 6-8 mg/day

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Total Iron requirement

Uterus, fetus, placenta	- 300 mg
Expansion of maternal RBC	- 500 mg
Daily loss through sweat, feces	- 200 mg
Blood loss during delivery	- 200 mg
	1200 mg

Iron saved by amenorrhea – 200 mg

Net requirement of Iron during pregnancy – 1000mg

Iron parameter:

00:18:10

- S Iron: decrease
- S ferritin: decrease
- Hepcidin: decrease
- Transferrin: increase
- TIBC: increase

Grading of Anemia

	WHO
Mild	9-10.9 gm%
Moderate	7-8.9
Severe	4-6.9
V. Severe	< 4

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Obstetric consequences of Anemia

00:22:20

Maternal	Fetal /neonatal
• preterm labour • puerperal sepsis • puerperal thromboembolism • poor tolerance to blood loss • postpartum depression	• prematurity • low birth weight / JUGR • poor mental / psychomotor performance • Low iron stores • Non reassuring fetal H.R • =Oligohydramnios (deceases AFI) • Fetal cerebral vasodilation • Fetal death

Diagnostic test:

- CBC (decrease RBC, increase WBC, increase platelets, decrease Hb, decrease Hct)
- Red cell indices (decrease MCV, decrease MCHC)

How to differentiate physiological /pathological

00:29:17



MCV - Normal



MCV - decrease

- Pap smear - microcytosis, Hypochromia
 - Anisocytosis, poikilocytosis
 - Malaria parasites
- Iron indices - decrease S. Iron
 - Decrease S. ferritin → most sensitive index for Iron Deficiency
 - Increase TIBC
- Stool examination - ova /cyst
- Urine microscopy - UTI / Renal failure
- Echo
- ECG
- Thyroid function test

Prophylaxis:

00:34:21

- Iron should be started with the 1st antenatal visit

WHO	When to start Iron	MOHEW (Indian Govt)
➤ 1st visit ➤ Elemental Iron - 60mg ferrous salt ➤ 400 mg of folic acid		- at 14 weeks - Elemental Iron - 100mg - 335 mg of ferrous sulphate 500 mcg ^Q of folic acid

Albendazole 400 mg
Or
Trimester
Mebendazoles 500 mg

} single dose in 2nd

Treatment of Iron deficiency anemia

1. Oral Iron
2. Parental Iron
3. Blood Transfusion

Oral Iron

- 1st line therapy
 - Treatment dose is 120 - 200 mg/day in 2-3 in divided doses
 - Ferrous sulphate
Ferrous fumarate
Ferrous gluconate
- } similar in their efficiencies

Enteric coated sustained release:

00:45:39

1. Carbonyl Iron
 2. Iron polymaltose complex
 3. Ferrous glycine sulphate
- } Expensive

Recommendations

1. Best taken on empty stomach – Early morning
2. Not to be given in food
3. Factors increasing iron absorption → vit c, Acid
4. Factors increasing Iron absorption → antacids, H₂ blocker & PPI Milk, & dairy products Calcium supplement Antibiotics (Quinolones)

Response to Iron therapy

00:50:30

- 1st response is increase in reticulocyte
- Occurs → 7-10 days after start of the therapy
- Rate of increase in Hb is 0.3 – 1 gm% /week → after only 2 weeks

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No improvement

- Compliance
- Hemorrhoids/worm infestation
- Folate deficiency
- Thalassemia
- Chronic illness

Parenteral Iron therapy

Intramuscular (IM)	Intravenous (IV)
1. Iron dextran (IM/IV)	1. Iron sucrose
2. Iron sorbitol citrate carboxy maltose	2. Ferric

Intramuscular (IM)

- 100 mg/day, Deep IM
- Z technique, to avoid skin staining
- Poorly absorbed
- Painful
- Stains the buttock
- Associated with gluteal sarcoma
- Anaphylaxis

IV preparations:

	Elemental Iron/ml	Maximum dose	Test dose
Iron dextran	50mg	100 mg	Required
Iron sucrose	20 mg	200 mg	only if allergies present
Ferric carboxy iron maltose	50mg	1000 mg	No

Calculation of dose:

01:03:51

- Total dose of Iron = $2.2 \times \text{Prepregnant weight (kg)} \times \text{Hb deficit} + 1000 \text{ mg}$

Or

$$= 0.3 \times \text{prepregnant wt} \times (\text{lb}) \times \text{Hb deficit} + 1000 \text{ mg}$$

$$\text{Hb deficit} = \text{Normal Hb} - \text{women's Hb}$$

$$(14\text{g/dl})$$

Indication for Parenteral therapy

1. Unresponsiveness to oral Iron
2. Non compliance
3. Intolerance
4. Close to delivery (Hb ≤ 7 & $9 \text{ gm \%}$ after 20 weeks

Rise in Hb with parenteral therapy is same as oral

Treatment algorithm:

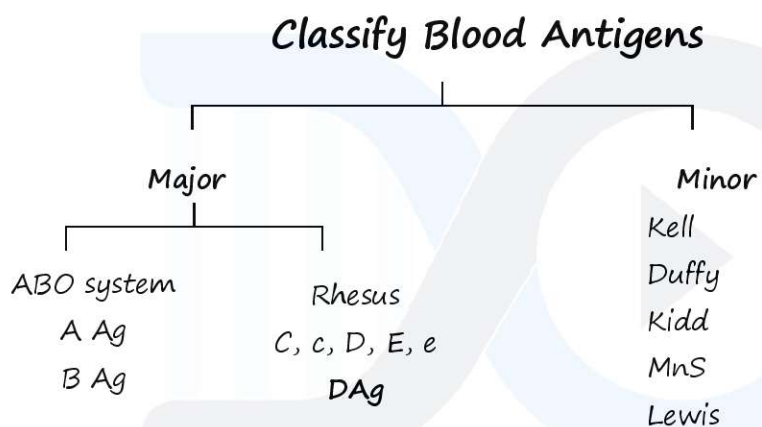
	Hb	≤ 20 wks	>20 wks
Mild	$9-10.9$ gm%	Oral	Oral
Moderate	$7-8.9$ gm%	Oral	Parenteral Iron
Severe	$4-6.9$ gm%	} Blood transfusion (any trimester)	
v. Severe	< 4 gm%		

RH-ALLO IMMUNISATION

- Prevalence of red cell antibodies in pregnancy -1%
- Prevalence of D-alloimmunisation (D - Ab) → 0.5 - 0.9%

Classify blood antigens

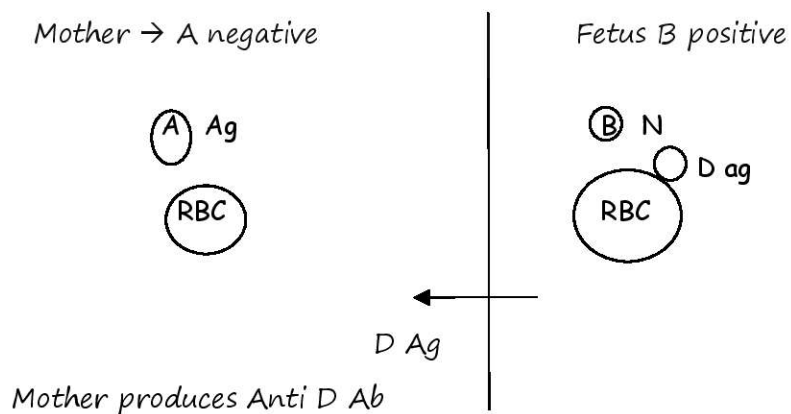
00:02:41



MOST COMMON AND MORE IMMUNOGENIC- KELL

ABO mismatch

00:13:54



When Fetal blood cross barrier

↓ Enter

Maternal Circulation

↓

Anti B Ab

↓ Attack B ag before WBC get sensitized
against D-Ag

Lyse Fetal RBC, before forming Anti D Ab.

- Other Rhesus Antigen
- Most common non-D allo immunisation → Anti - E
Most severe non-D allo immunisation → Anti-c

Effects of ABO system AfraTafreeh.com 00:13:54

- ABO incompatibility protects against Rh sensitisation

Risk of Sensitisation

With ABO

Compatibility

16%

Mother - A negative

Baby - A positive

ABO Incompatibility

2%

Mother → A negative

Baby → B positive

Alloimmunisation in minor antigens

00:13:54

- Most common minor antigen causing sensitisation → Kell
- Other potential antigens causing allo immunisation → Duffy
 - Fya
 - Kidd
 - Jka
 - MNS
- No risk of alloimmunisation
 - Lewis Antigen Le^a , Le^b
 - I antigen
 - Duffy Fy^b
- Most of Rhesus Alloimmunisation → during pregnancy
- 90% of Kell alloimmunisation → due to blood transfusion
- sReaction is {severe rapids} → Destroys precursor RBCs in bone marrow leads to severe Anemia.

LOGIN FOR SUCCESS

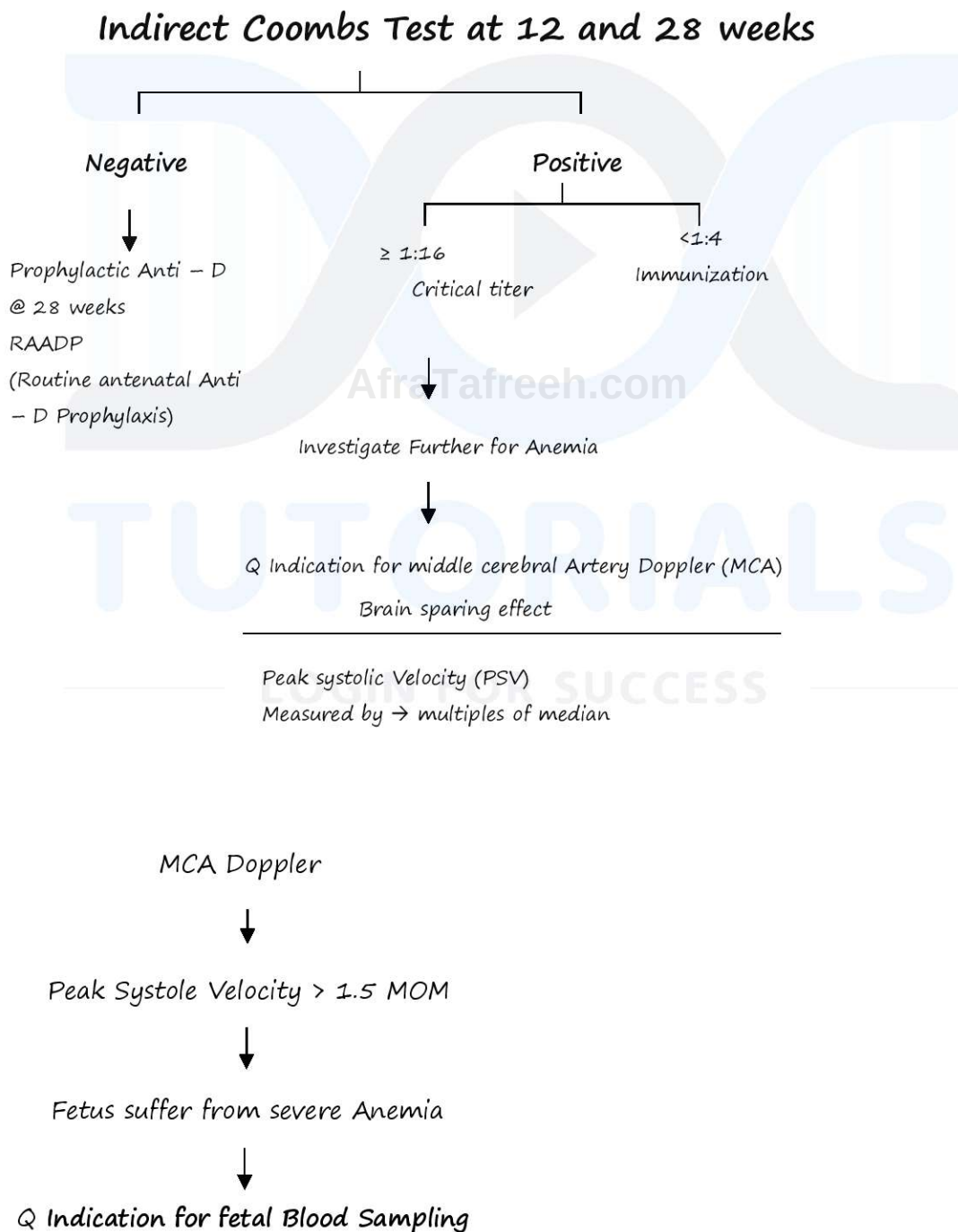
Causes of Feta-maternal hemorrhage

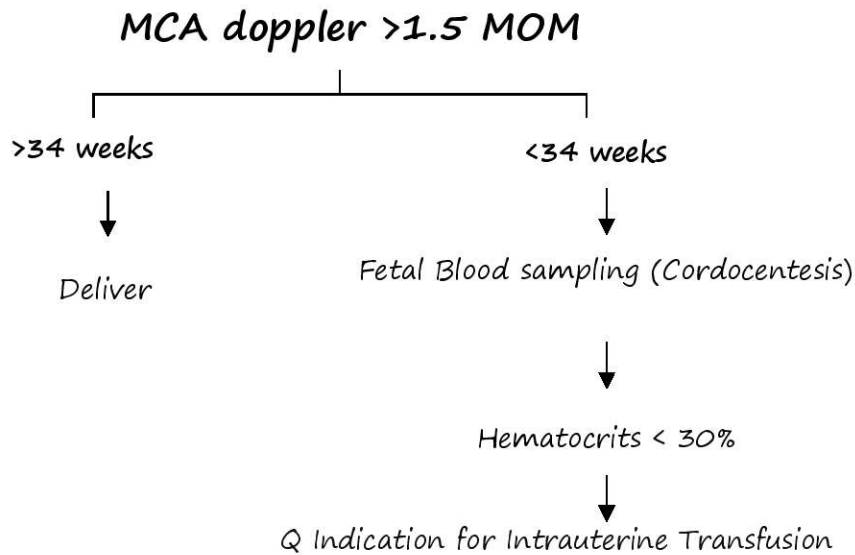
00:24:38

- Potential sensitizing events that lead to R cell alloimmunisation
 1. Minimum amount of blood required to sensitive → 0.1ml
 2. 90% of Rhesus alloimmunisation occurs during – delivery
 3. Sensibilisation

- Condition where exposure occurs during 1st pregnancy & sensible number of antibodies seen in next pregnancy
4. Rhesus system coded by chromosome - 1
5. Incidence of sensitization with Anti-D - $< 0.2\%$

How to screen Rh negative mother





- Usual Feto-maternal Haemorrhage → <15ml of fetal RBC or <30ml of fetal blood
 - 300mcg (or) 1500 IU – Neutraliser 15ml of RBC
 - 10mcg – Neutraliser – 1ml of fetal blood
 - Prophylactic dose (RAADP) given at 28wks for non-sensitised
 - 2nd dose given within 72hrs post-partum
 - Can ICT positive after Anti-D – yes
 - Can DCT positive after Anti - D – Yes
- } Weak positive

How to Quantify dosage

00:17:46

- Qualitative Test – Rosette Test
- Quantitative Test – Kleihauer Betke Test ^Q / Flow cytometry
- For Every 1ml of fetal blood → 10µg Anti D – Required

Estimated fetomaternal hemorrhage

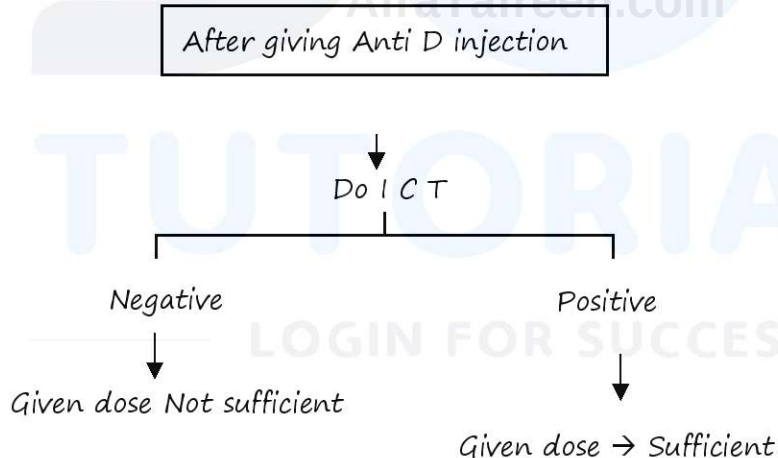
Material blood volume (in ml) x Haematocrit x % fetal cells in KB (kleiheur betke)

Newborn Hematocrit

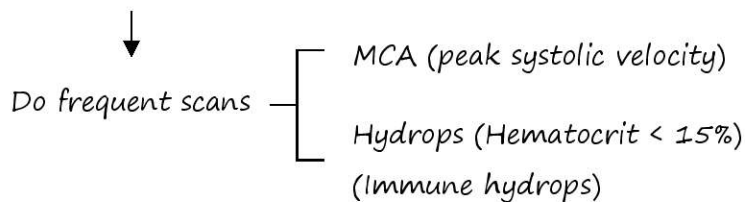
Dosage of Anti D

- IM (Intra Muscular) deltoid → Maximum dose – 1500mcg
 - 5 doses of 300mcg each in 24 hrs
- IV (Intravenous) → Maximum dose – 1800 mcg
 - 3 doses of 600 mcg each at 8 hrs gap

To check whether dose enough / not



- If women are already sensitised



Order of Management

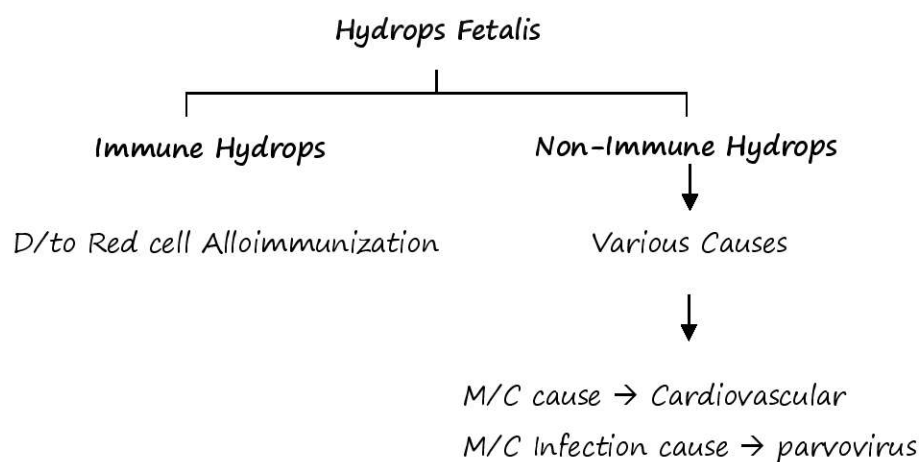
- ICT titre → MCA doppler → Culdocentesis fetal blood sampling → Intra uterine Blood Transfusion

Hydrops Fetalis

- Definition:
2/ more effusion (pleural / pericardial effusion / Ascites)

(or)

1 effusion + Anasarca (Generalised edema)



Intra uterine Transfusion

- Volume to be transfused = Estimated fetal weight in gram $\times 0.02$ for each 10% increase in Haematocrit
- Target Haematocrit $\rightarrow 40-50\%$
- Example:
 - Hydrops Fetus with Haematocrit $\rightarrow 20\%$
If EFW = 1000
 - Present 20% $\xrightarrow{\text{Increased by } 30\%}$ Target 50%
 - Therefore, volume calculated by = $1000 \times 0.02 \times 3$
- Q Following Intra uterine Transfusion Haematocrit drops by 1%/day

Mirror Syndrome

- Both mother & baby mimick each other
- Also known as – **Ballantyne syndrome**

Causes of mirror Syndrome

- D – Alloimmunisation
- TTTS (Twins to Twin Transfusion Syndrome)
- Placental chorioangioma
- Cystic Hygroma
- Ebsteins anomaly
- Sacrococcygeal teratoma
- Chylothorax
- Bladder outlet obstruction
- Supra Ventricular Tachycardia (SVT)
- Vein of galen malformation
- Congenital Infection
- Pre – Eclampsia

RH NEGATIVE MOTHER

Quantification of Fetomaternal Haemorrhage(FMH):

- Kleiheur betke test (Massive FMH) within 2 hours of delivery
- 1500IU/300mcg neutralize
 - 30ml of fetal blood
 - 15ml of fetal RBC
- 99% of Rh negative women has FMH <4ml

Condition Associated with Large FMH 00:04:13

- Traumatic delivery (CS, foreps)
- Manual removal of placenta
- Still birth
- Fetal defects
- Abdominal trauma
- Twin pregnancies @ delivery
- Unexplained hydrops fetalis

Standard Dose:

- 1500IU
- 1% will have FMH > 4ml, 0.3% FMH > 15ml
- 99% will have FMH < 4ml
- Minimum dose – 500IU (1% uncovered ,99% covered)

- Standard dose – 1500IU neutralise 15ml of fetal RBC
- 500 IU – 4ml
- 1500IU – 15ml
- Other Tests
 - i. Flow cytometry
 - More accurate
 - Indicated only when KB shows large FMH
 - ii. Rosetting technique
 - KB test (test of choice)
 - 500IU will neutralize 4ml of FMH
 - 125IU will neutralize 1ml of FMH
- Each additional ml of FMH – 125 IU of anti D
 - 1500IU → 15ml FMH
 - ? → 20ml FMH
 - $5\text{ml} \times 125 = 625\text{IU} + 1500\text{IU}$
 - $\frac{5\text{ml} + 15\text{ml}}{20\text{ml}}$
 - 125IU for every ml
 - Minimum dose
 - <20weeks – 250IU
 - 20weeks & above – 500IU
 - Standard dose – 1500IU

Timing and Site

- As soon as sensitizing event occurs, ideally within 72hours

- Can be given upto 10days
- Ideal site – deltoid (I.M)
- Can be S.C/I.V
- Bleeding disorders → I.V
- Consent to be taken & documented

Miscarriagee

- Miscarriage
 - Spontaneous miscarriage/before 12weeks/no instrumentation, in this condition where anti-D is not required
 - Complete abortion where anti -D is not required.

2nd Trimester Abortion

- Anti -D has to be given
- Surgical evacuation – Yes
- Induced abortion/MTP – Yes
- Incomplete aboetion → Yes

Threatened Miscarriage:

- After 12 weeks – Yes
- Continuous bleeding after 12 weeks – Given anti – D @ 6 weekly interval
- Mild bleeding after 12weeks which stopped – Avoid anti – D
- Repeat bleeding before 12weeks → consider – Yes

Ectopic Pregnancy & MTP:

- Anti – D should be given
- Regardless of GA & Rx

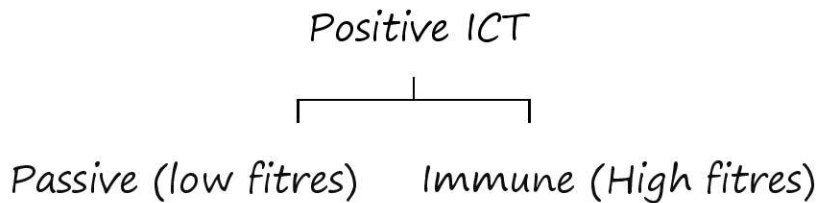
Sensitizing Events:

- Invasive prenatal diagnosis (CVS/amniocentesis/cordocentesis/IuT)
- Other invasive procedure (Laser/embryo reduction/insertion of shunt)
- Antepartum haemorrhage
- ECV (including attempted)
- Any abdominal trauma
- Fetal death
- Recurrent vaginal bleeding after 20 weeks
 - Anti – D 500IU + KB 2 weekly repeat anti- D 6 weekly interval
 - If GA
 - < 20 weeks – 250IU
 - $\geq$ 20 weeks – 500IU + KB test

Routine Antenatal Anti- D Prophylaxis (RAADP):

- ICT @ booking visit 28 weeks
- ICT positive → No use of RAADP
- Dose
 - 2 dose → 500IU @ 28 & 34 weeks
 - 1 dose → 1500IU @ 28 weeks

- Sensitizing events: 250/500 IU – regardless of RAADP
- Anti – D persists in circulation for 8 weeks in circulation whenever in doubt about ICT → give anti – D



- Passive immunization and DCT → positive



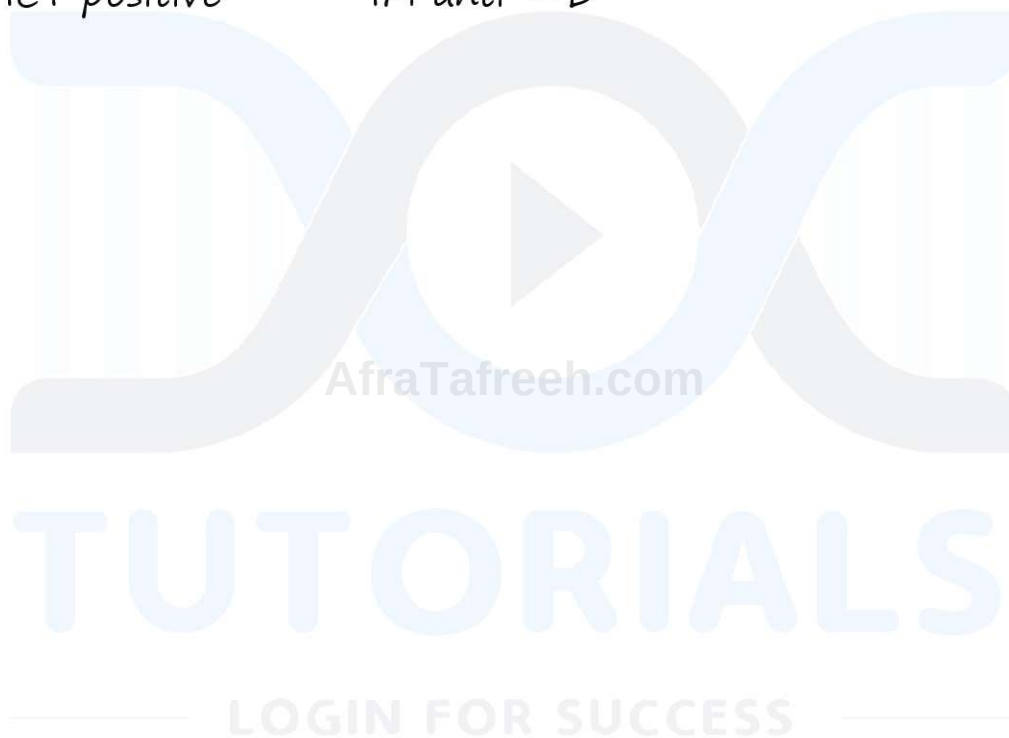
- Passive immunization & fetal haemolysis → NO (RAADP)
- Anti-D – 28 weeks
- ICT → Yes
- DCT → Yes
- Fetal hemolysis → No
- If RAADP is declined
 - ICT @ booking @ 28 weeks

Mismatched Platelet Transfusion:

- Contaminated with red cell (blood group Ag)
- Opositive plt → 'O' neg lady – sensitized/anti – D Ab
- Anti – D 250IU for every 3 unit platelet transfusion

Mismatched Blood Transfusion:

- KB test
- 500IU = 4ml. FMH
- 125 IU = 1ml FMH
- > 15ml FMH → 1500 IU
2500IU
- IV anti - D → twice as effective as
ICT positive IM anti - D



VARICELLA IN PREGNANCY

- Teratogenic Virus, incubation period 2-3 weeks
- Infectious period begins 2 days before onset of rash and 5 days after onset of rash until the lesions crust completely

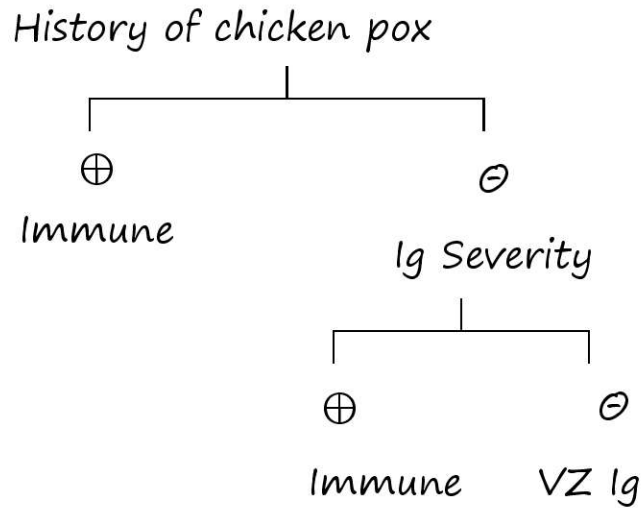
Routine Antenatal Screening:

00:01:42

- Not recommended
VZ Ig ← Negative
- Susceptible for infection
- Vaccination during pregnancy → C/I absolutely because it is a live vaccine (attenuated)
- Post partum period → vaccinated during this period
- Inadvertently → Not an indication for MTP

Prevention During Pregnancy 2 doses:

- Wait 4 weeks before last dose and pregnancy
- Avoid exposure
- Pregnant women to chickenpox/ zoster
 - >90% of women immune to chicken pox



Infectious Period After Exposure:

00:09:53

- 8-28 days exposure VZIG ⊕
- 8-21 days after exposure VZIG -

Second Dosed VZIG:

- VZIG has to be given within 10 days of exposure
- 2nd dose → 3 weeks after 1st dose

Maternal Risks of Varicella:

- Pneumonia
- Hepatitis
- 2^o bacterial infections
- Sepsis
- Mortality

Rx of women who develop chickenpox: 00:13:35

- Oral acyclovir – 800mg 5 times a day * 7 days
- Within in 24hours from onset of lesion
- VZIG – Only for exposed women with no lesion upto 10days from exposure
- IV acydovir is for severe disease
- Role of VZIG in women with rashes



No role

Need for hospital admission



Severe disease

- Mode of delivers → vaginal/cs
- Time of delivery

14 days



- If develops chickenpox in last 7 days of pregnancy or within 7 days postpartum



Neonatal varicella

- Risk of miscarriage – Nil

Risk of fetal Varicella Syndrome (FVS): 00:19:40

chorioretinitis

placental calcifications

microcephaly

cerebral calcifications

neurological abnormality

mental retardation

hypoplasia of limb

microphthalmia

cataract

- It is highest in 2nd trimester (0.9–1%)
- In 1st trimester (0.4%)

Routes of fetal/neonatal infection:

- Transplacental
- Vagina ascending infection
- Direct exposure to lesion during delivery

Rates of Neonatal Infection:

- If mother develops lesion 1–4 weeks before delivery
→ 50% incidence of clinical varicella
↓
27% of this 50%

Indication for VZIG in Neonate:

- 7 days before → 7 days after delivery
- If in 14 d develops lesion
- Recommendation on breast feeding – Yes not contraindicated
- If she has active lesion close to nipple
↓
Express the milk from breast until lesion starts crusting

MALARIA IN PREGNANCY

Fetal Complications:

00:00:38

- Miscarriage
- Still birth/IUD
- Preterm labour
- Low birth weight
- Congenital malaria (<5%)

Maternal Complications:

- Anemia (Hb<8g%)
- DIC/PE
- Thrombocytopenia
- convulsions
- Hypoglycemia (<2.2mmol/lit)
- circulatory collapse/shock
- Metabolic acidosis (PH<7.3)
- Jaundice
- Renal failure (creat > 265mmol/PH)
 - U/O < 0.4 ml/kg /hr)
- Pregnancy – increase risk of mosquito bite
 - Increase severity of disease
 - Mortality increase with pregnancy
 - Fever /flu like illness upto 1year
- Components of malarial prevention

A-Awareness

B- Bite protection

C- Chemoprophylaxis

D-Prompt diagnosis & treatment

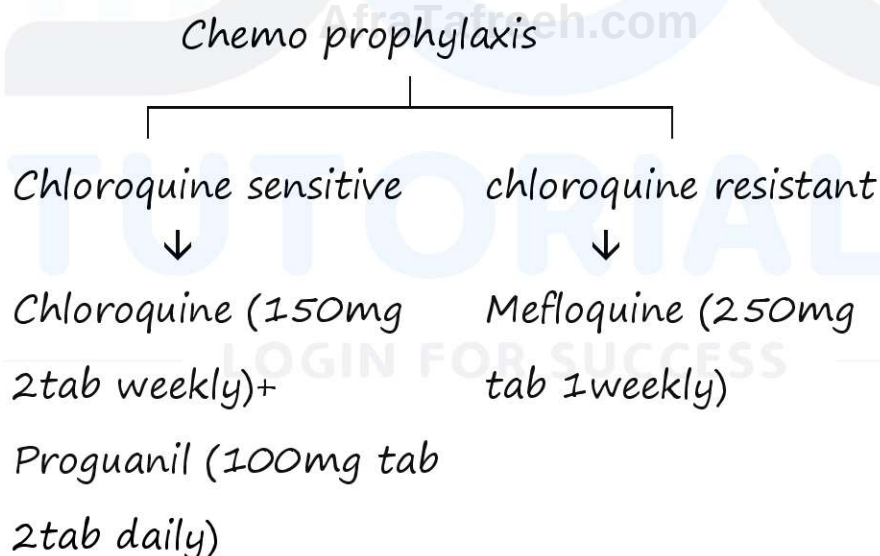
Bite Prevention:

00:10:15

- i. Repellants (50% DEET)
- ii. Pyrethroid & permethrin sprays
- iii. Pyrethroid impregnated bed nets
- iv. Pyrethroid impregnated clothes
- v. Electrically heated bats

Chemoprophylaxis:

- It is not 100% effective



C/I of Mefloquine:

- i. Seizure disorder
- ii. Neuropsychiatric disorder
- iii. Depression
- iv. Allergy to Mefloquine

- In this case DOC is atovaquone 250mg
 +
 Proguanil 100mg } 1tab/day

- Proguanil → 5mg folic acid (prophylactic) for pregnant / seeking to become pregnant
- All these drugs are safe and used in all trimesters and can be used in lactating mother also

Clinical in Pregnancy:

- i. Doxycycline – Cause teeth discoloration, congenital cataract
- ii. Primaquine – Hemolysis especially in G6PD deficiency

Standby Treatment in Pregnancy:

- Quinine 600mg TID + clindamycin 450mg TID * 7 days
- 2nd choice
 - Atovaquone + Proguanil
- Should be started within 24 hours of symptoms

MULTIFETAL PREGNANCY – I (TYPES & RISK FACTORS)

Definition

00:00:25

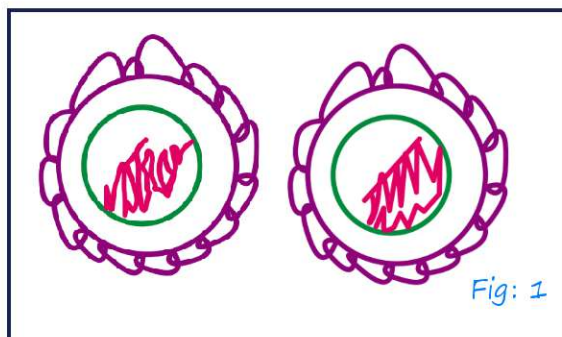
- Simultaneous occurrence of more than 1 fetus utero is multiple pregnancy.
- If they are two in number
 - Twins – 97%
 - Triplets
 - Quadtriplets

Types of twin pregnancy

- i. Dizygotic twins/fraternal twins.
- ii. Monozygotic twin/identical twins.

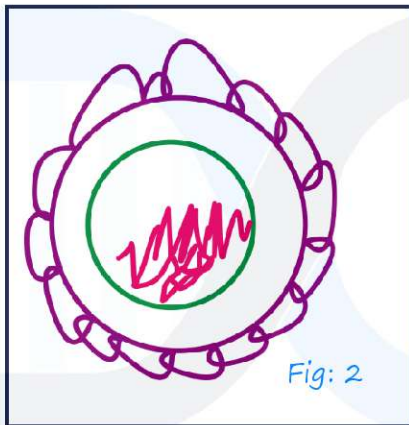
Dizygotic

- Two different oocytes to which two different sperm fuse as a result we get two different embryos.
- These embryos' will have their own chorions & amnion and also have their own inner cell mass.
- Dizygotic twins are always Dichorionic, Diamniotic



Monozygotic twinning

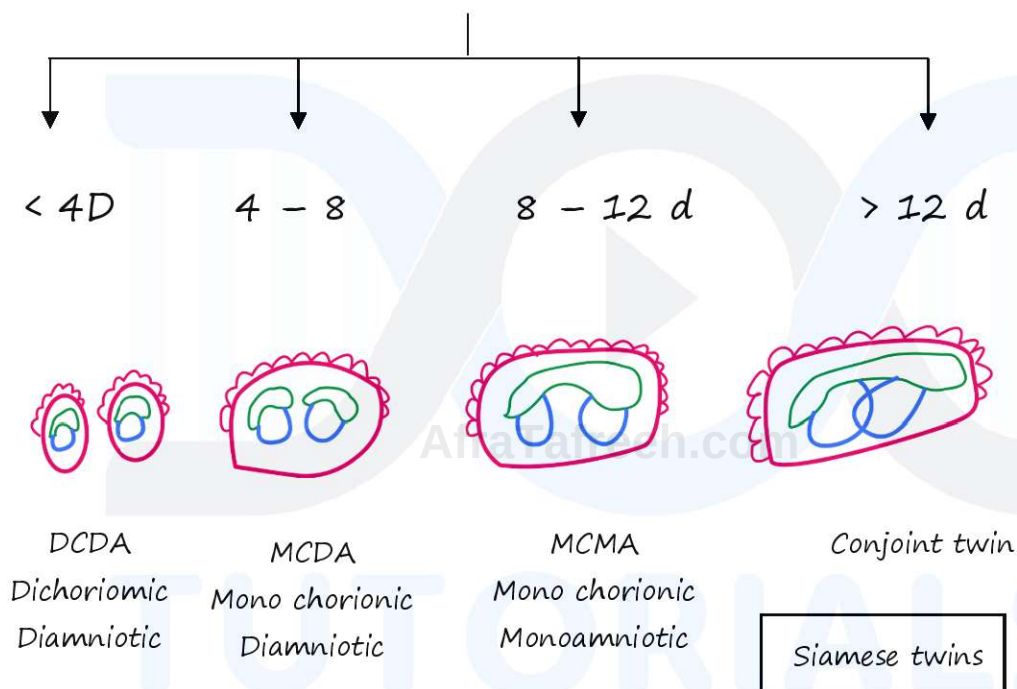
- It is one single oocyte forming one single sperm forming one single embryo.
- It has one chorion, one amnion and inner cell mass.
- Monozygotic twinning occurs purely by chance where the single fertilized over either divides into two in zygote stage or blastocyst stage.



- If the division occurs within the first four days – Chorion will divide completely and amnion will also divide. Therefore, there will be two different chorions, two amnion and two fetuses. So, it is called as the dichorionic and diamniotic (DC DA).
- If the division occur between 4th & 8th day chorion cannot divide anymore, amnion divides it will be a single chorion with two amnions and two embryos on fetuses and it is called as mono chorionic Di amniotic (MNDA).
- If the divisions occur between 8-12 neither the chorion or amnion divides anymore. So, there will be

single chorion and amnion having two separate foetus not attached to each other in this case we call it Monochorionic Monoamniotic (MC MA).

- If the division occurs beyond 12 days we have single chorion inside with single amnion to which attached two fetuses which are joined to each other. This twinning is called as conjoint twin or Siamese twin.



- Dizygotic only has risk factors applicable only for this
- Monozygotic purely occurs by chance
- Incidence of dizygotic twin is – 1 in 80.
- Incidence of monozygotic twin is 1 in 250
- MCDA is the most common monozygotic twin – 75%
- 24 to 25% are DCDA monozygotic twin.
- MC MA and conjoint together make less than 1%.
- Overall, most common type of chorionicity – DCDA
- Overall, most common type of zygosity – Dizygotic

- Most common type of chorionicity in monozygote – MCDA
- Super fecundation – Dizygotic twin ova/sperm → 2 in number (2+2) released in the same cycle.
- Occurs in 2 different coitus
- Selper fetation – Dizygotic twin ova/sperm → 2+2
- The two oocytes released in fertilization are released in two different cycle.
- Occurs in 2 different coitus

Risk factors

- Only applicable for dizygotic twin
 - 1.Black women
 - 2.Nigerian (1 in 20)
 - 3.Advanced maternal age
 - 4.Advanced paternal age
 - 5.Increasing parity
 - 6.Maternal family history
 - 7.Paternal family history
 - 8.Obesity
 - 9.Ovulation induction drugs.
 10. ART (artificial reproduction techniques)
- More common in female foetus than male foetuses.

MULTIPLE PREGNANCY – II (DIAGNOSIS OF TWIN PREGNANCY)

Diagnosis of Twin Pregnancy

00:00:15

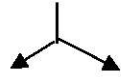
- Chorioncity is more important than zygotity because chorioncity only determines the outcomes of twin pregnancy
- More chorion, better prognosis e.g., Dichorion Diamniotic has better prognosis

	Gender	Blood Group	DNA/HLA	Fingerprints	Placenta
Monozygotic Twins	Always same there for they share same genetic material	Always same	Always same	Always different	Mono OR Dichorionic
Dizygotic Twins	Same / Different	Same / Different	Always different	Always different	Dichorionic

- Exception: In Monozygotic twins, where genders it different Karyotype – 47xxy, Monozygotic twin

There was post zygotic loss (After division of zygote)

One twin lose x Other twin lost y



46XY 46xx → Female fetus



Becomes male fetus

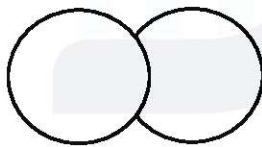
OR

There can be 46xx/ 46xy mosaic



Known as mixed gonadal dysgenesis

- Dizygotic always has dichorionic placenta
- Exception: Fusion of placenta in dizygotic
 - They appear monochorionic



Monochorionic placenta

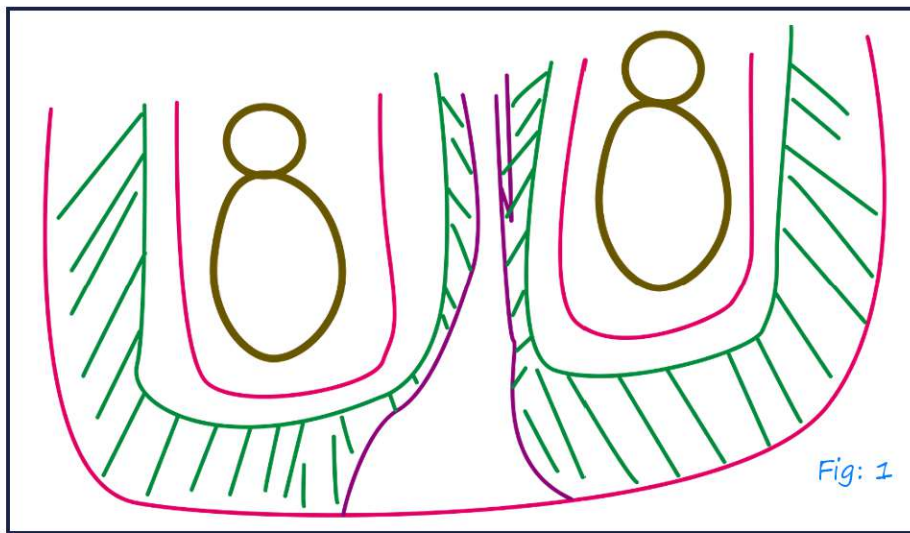
USG

00:10:14

- Ideal time < 14 wko to see chorionicity
- Normally we see at 12 weeks

Dichorionic Diamniotic (DCDA) twin:

- Between the 2 placenta there is inverted y shaped area is known as λ sign

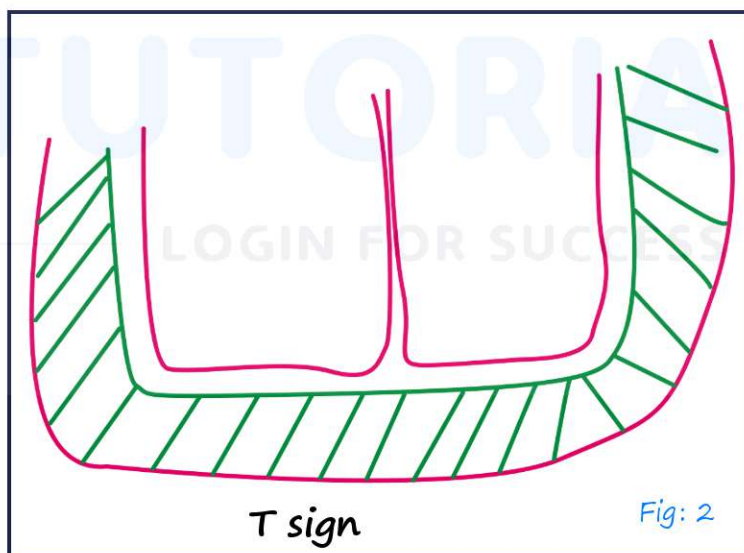


λ lambda sign (or) twin peak sign (or) Delta sign

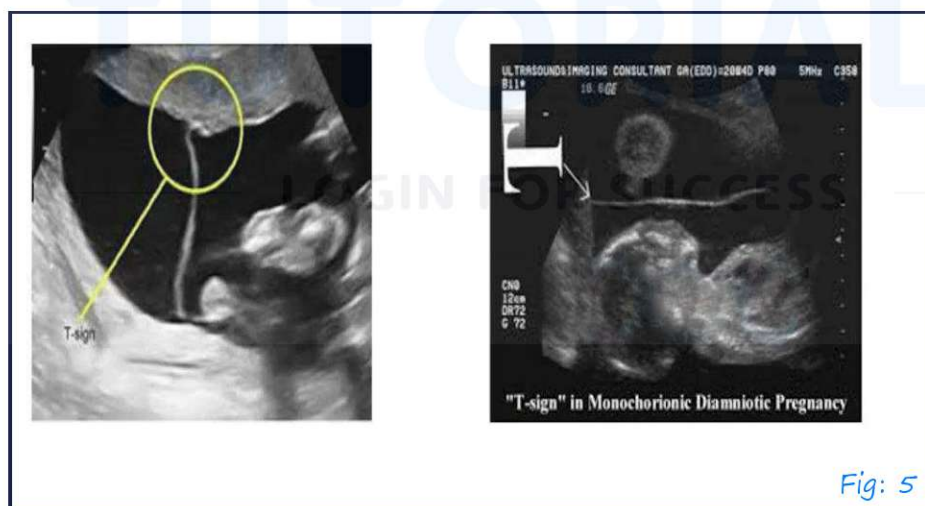
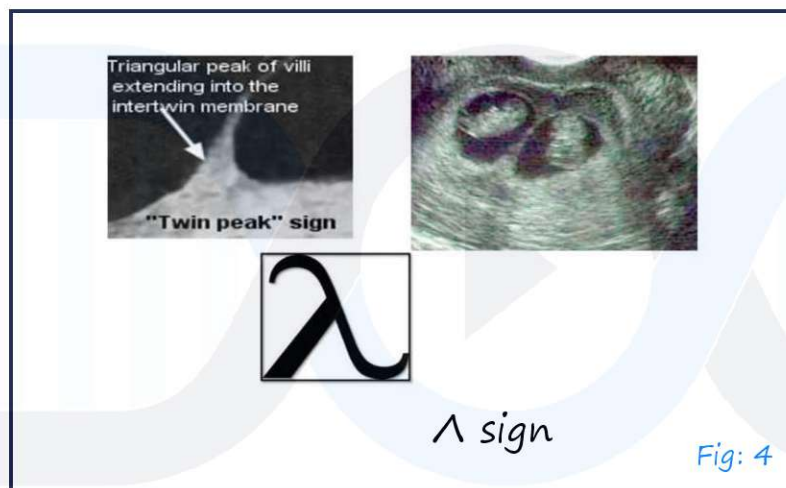
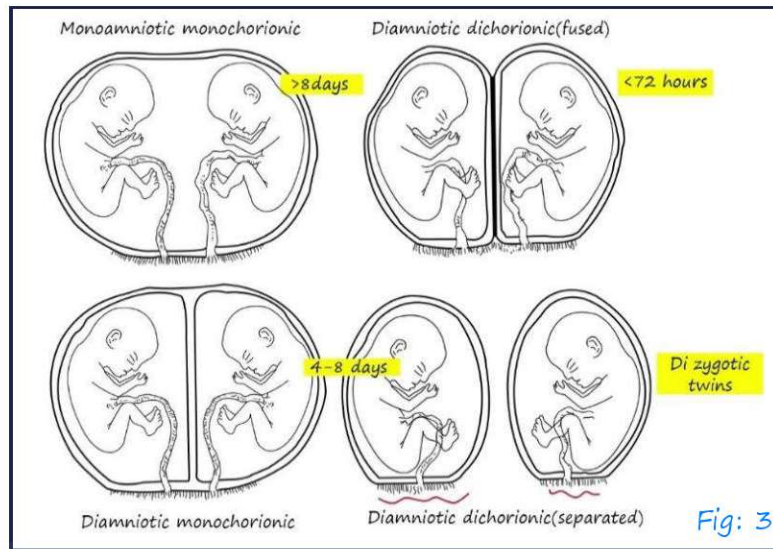
- Order of Membranes Between the 2 fetuses : Amnion, Chorion, Chorion, Amnion (ACCA)
- Thickness of membrane ≥ 2 mm

MCDA twin

- T sign is seen



- Order of membranes between 2 fetures; Amnion – Amnion (A-A)
- Thickness of membrane < 2 mm



- Thickness of intervening membrane >2mm suggest dichorionicity
- 1st image: T sign



Fig: 6

Trichorionic triamniotic

Question 1

Multiple pregnancies are MC seen with

- a. T. clomiphene (5-10%)
- b. T. Clomiphene & dexamethasone
- c. T. Dexamethasone
- d. Pulsatile GnRH (20-40%)

AfraTafreeh.com

Answer: d

Question 2

Earliest method to determine the zygosity is

- a. Blood grouping
- b. Sex of fetus
- c. Placenta examination
- d. Amniotic fluid

LOGIN FOR SUCCESS

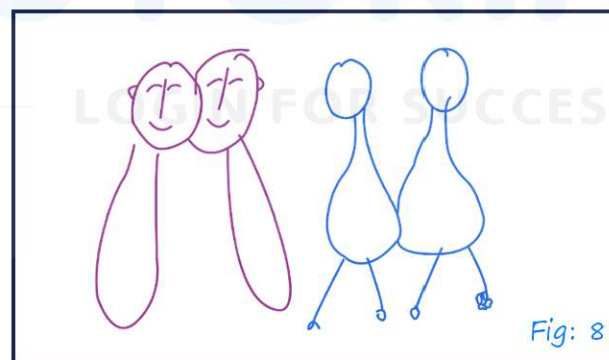
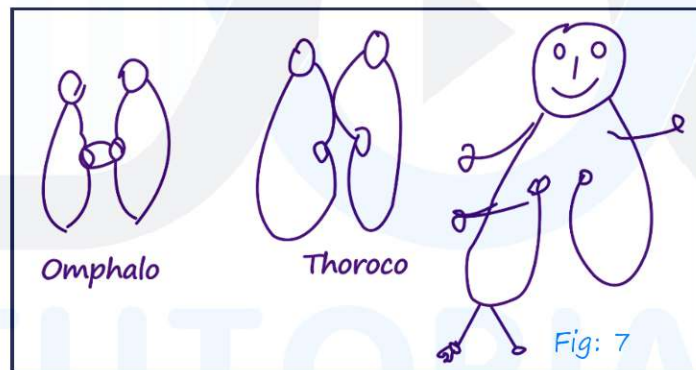
Answer: C

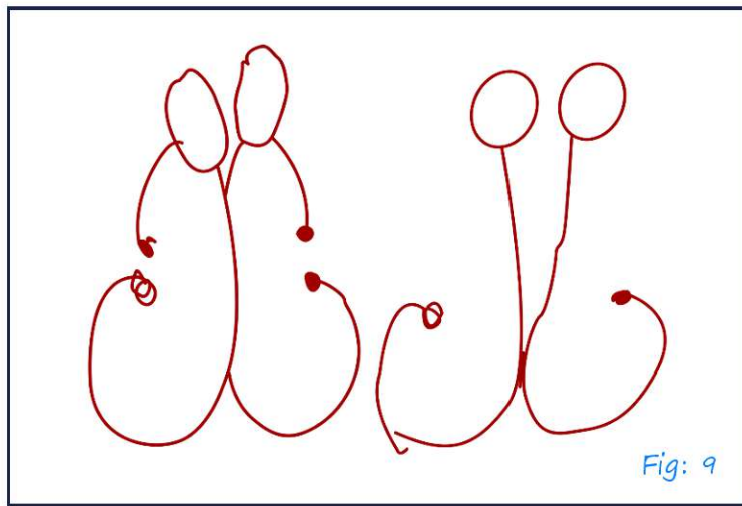
Conjoint Twins

- Incidence: 1 in 1 lakh

Types

Types	Attachment Point
i. Omphalopagus	Umbilicus
ii. Thoracopagus	Chest to chest
iii. Cephalopagus	1 head with 2 trunk
iv. Craniopagus	2 different heads
v. Ischiopagus	Pelvic bones attached side to side
vi. Rachiopagus	Back to back (entire spine)
vii. Pyopagus	Buttock to buttock





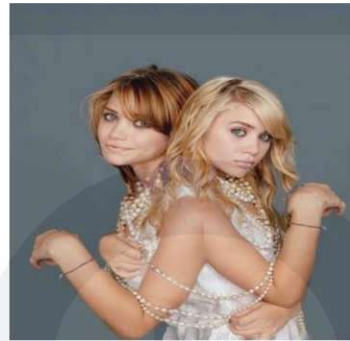
- Thoracopagus



- Cephalopagus / syncephalus



Omphalopagus Fig: 12



Rachipagus Fig: 13



craniopagus Fig: 14



Fig: 15

Rachipagus

- MC type of conjoin twin is Thoracophagus

MULTIFETAL PREGNANCY- IV (MANAGEMENT OF TWIN PREGNANCY)

Twin discordancy

00:00:15

- Difference in birth weight of more than 20% with larger twin's weight

Formula $\frac{\text{Wet of larger twin} - \text{wet of smaller twin}}{\text{Wet of larger twin}} \times 100$

- It the value is > 20% It is called twin discordancy
- 2nd definition -Difference in abdominal circumference of > 20mm
- Causes of discordancy → difference in growth potential, or difference in genders or, avascular anastomosis
- Discordance twin should be delivered vaginally only if bigger twin is in the presenting area

Single twin demise

- It can occur in early pregnancy or late pregnancy
- Most commonly seen in monochromic pregnancy
- When the single twin demise ours in early pregnancy
 - either one twin disappears, it is called as vanishing twin, or else it gets minified and struck

to the placenta which is known as fetus papyraceus/
fetus compresses

- If the dead twin disappears it is called as vanishing twin
- If one twin dies, it becomes dangerous for the twin, as is advanced pregnancies single twin demise is due to vascular anastomosis
- In the dead twin, there is a sudden fall in resistance
- Blood from the living twin flows into the low-pressure area. due to which sudden hypotension occurs which leads to neurological damage
- Emboli of thrombotic material from dead twin can cause neurological damage in the living twin

Management of single twin demise

00:07:45

	Monochorionic	Dichorionic
1 st trimester	Continue the pregnancy	Continue the pregnancy
Early second trimester	Terminate	Continue
Late 2 nd Early trimester	Continue & deliver by 34 to 37 weeks	Deliver at term

How to present prematurity

- Bed rest
- Increase Oncolytic, pessary cachage, progesterone is not used
- It depends on DCDA to deliver by 38 weeks
 - MCDA by 34-37 weeks (36 weeks)
 - MCMA → 32-34 weeks (34 weeks)

Route of delivery

00:12:54

- Cephalic combination (m/c)
- Cephalic breech
- Cephalic transverse

Cephalic combination

- Both can be delivered vaginal

Cephalic breech

- The first one can be delivered vaginal & second one assisted breech delivery or LSCS

Cephalic transverse

- First one can be delivered vaginal and second one internal public version

Indications for C Section

1. First twin non cephalic
2. Twins with previous 2 or more LSCS
(previous/LSCS - Trial of vaginal delivery)
3. Monoamniotic twins



FETAL ANOMALIES IN PREGNANCY AND PRENATAL TESTING

Age related risk of downs at mid trimester

	Singleton	Twin
35years	1 in 250	1 in 125
40years	1 in 69	1 in 37
45years	1 in 19	

First trimester screening

00:01:27

- Triad of 3 components
 - Nuchal translucency
 - PAPP-A (Pregnancy Associated Plasma Protein A)

	T_{21}	T_{18}	T_{13}
Nuchal	Increase	Increase	Increase
HCG	Increase	Decrease	Decrease
PAPP A	Decrease	Decrease	Decrease

Nuchal translucency

- Maximum thickness of subcutaneous translucent area between skin and soft tissue overlying fetal spine
- To measure NT → Baby should be in mid sagittal plane



- Ideal gestational age to measure NT – 11-14weeks
- Ideal CRL to measure NT – 38-84mm
- Differential diagnosis → Cystic hygroma
- Cut off → 3mm
- >3mm NT
 - Increase risk of aneuploidies
 - Increase risk of genetic syndromes
 - Increase birth defects
 - Increase cardiac anomalies
- NT >3mm
 - Indication for invasive testing/cell free DNA, fetal echo
- Detection rate with NT alone → 64-70%^Q
- Detection rate with NT+HCG+PAPPA (First trimester screening) → 80-84%^Q
- Detection rate is higher is performed @11weeks > 14weeks

PAPP-A

00:09:58

- Low PAPP-A <5th centile
- Low PAPP-A seen in

- i. Preterm birth
- ii. IUGR
- iii. Pre-eclampsia
- iv. IUD

Second Trimester Aneuploidy Screening (QUAD)

- Includes → α FP, unconjugated estriol, HCG, Inhibin
- A/K/A quadruple test
- Ideal gestational age: 15-21 weeks

	T_{21}	T_{18}	T_{13}
α FP	Decrease	Decrease	Normal
Unconjugated estriol	Decrease	Decrease	No data
HCG	Increase	Decrease	Decrease
Inhibin	Increase	-	-

- DOWNS → High → HCG, Inhibin

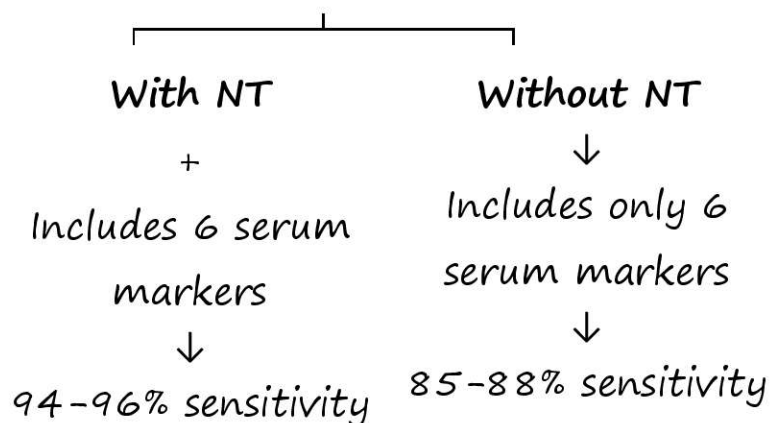
Integrated screening

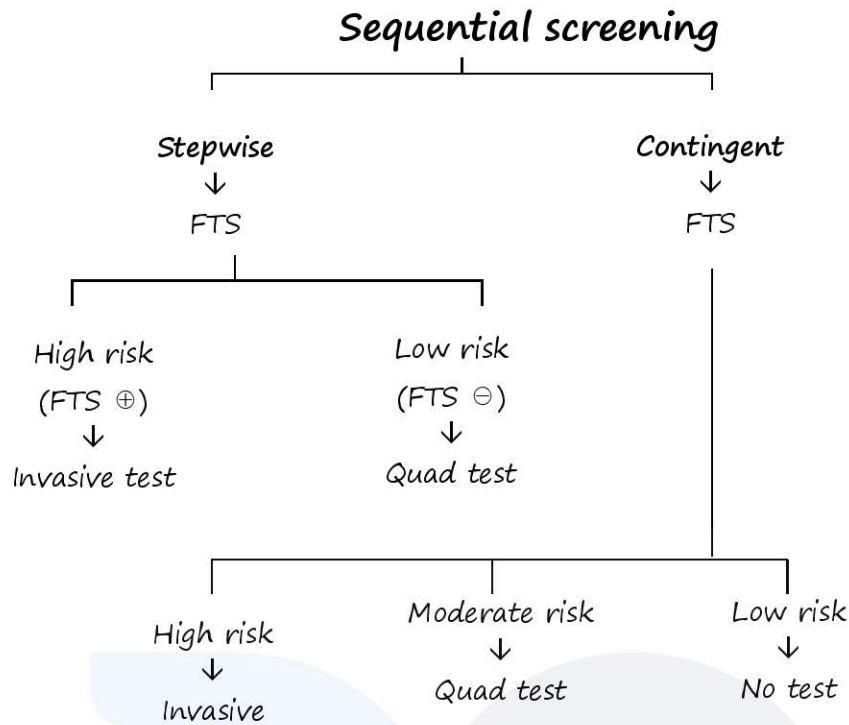
00:14:01

First trimester screening

+

QUAD





- Detection rates with various techniques:

	<i>T₂₁</i> detection rate
First trimester screening	80 - 84%
NT alone	64-70%
Quadruple screening	80 - 82%
Integrated screening	94 - 96%
Sequential screening	92 - 91%
Cell free DNA	99%

- Most sensitive screening test → cell free DNA.

Cell free DNA test:

00:20:59

- Screening test
- Also known as Non-invasive prenatal testing (NIPT)
- Testing DNA fragments that derived from apoptotic trophoblast.
- Can be performed any time after 9-10 wks. GA
- Not gestational age dependent.
 - Sensitivity of cell free DNA:
 - (T₂₁) Downs – 99%
 - (T₁₈) Edward – 96%
 - (T₁₃) Patau – 91%
 - Turners – 90%

Indications

1. Maternal age > 35yrs
2. First trimester screening or QUAD test screening – positive
3. USG showing minor aneuploidy marker
4. Past pregnancy with Trisomy
5. Either parent carrying balanced translocation (Chr21)

Indications

- i. May not always reflect fetal DNA

Possible contaminations of → Maternal malignancy

Mosaicism/placental mosaicism

Early demise of Twin.

- ii. Not recommended for twin pregnancy

iii. Assay failure
(or)
No call results

is 4-8% → Indication for amniocentesis

Confirmatory tests

- Karyotyping
- Amniocentesis
Cordocentesis
Chorionic villous sampling (CVS)

Ultrasound soft markers of Aneuploidy 00:27:10

- Aberrant right subclavian artery
- Brachycephaly or shortened frontal lobe
- Echogenic bowel
- Flat facies
- Echogenic intracardiac focus
- Nasal bone absence or hypoplasia
- Nuchal fold thickening
- Renal pelvis dilation (mild)
- Sandal gap between 1st & 2nd toes
- Shortened ear length
- Single transverse palmar crease
- Single umbilical artery
- Short femur
- Short humerus
- Widened iliac angle

HEART DISEASE IN PREGNANCY

Cardiovascular changes during Pregnancy

- ↑ HR/↑ PR – 10 bpm
- ↑ Cardiac output
- Edema
- Fatigue/Exercise intolerance
- Jugular venous distension (JVP ↑)
- Loud & widely split S_1
- Split S_2
- Occasional S_3
- Systolic murmurs
- ECG – (Lt) axis deviation
- Increased cardiac silhouette
- Shift in apex bat

Clinical indicators of heart disease during pregnancy

symptoms

- Progressive dyspnea /orthopnea
- Nocturnal cough
- Hemoptysis
- Syncope
- Chest pain

Clinical features

00:04:07

- Cyanosis
- Clubbing of fingers
- Persistent neck vein distension
- Systolic murmur $\geq$ grade 3
- Diastolic murmurs
- Cardiomegaly
- Persistent tachycardia
- Arrhythmias
- Persistent split S_2
- Presence of S_4
- Pulmonary hypertension

Classification of functional heart disease

- Class I – Uncompromised, No limitation of physical activity, no symptoms
- Class II – Slight limitation of activity, Discomfort with ordinary activity
- Class III – Marked limitation of activity, Discomfort with less than ordinary activity
- Class IV – Severely compromised, discomfort/Angina at rest. Inability to perform any activity

Predictors of cardiac complications [NOPE criteria]

00:07:29

- It indicates severity and outcome of heart disease in pregnancy
 - N – NYHA class III or IV
 - O – Left Heart obstruction
 - P – Prior heart failure/stroke/arrhythmia
 - E – Ejection fraction < 40%

Contraindications for pregnancy [WHO criteria IV]

00:10:17

- i. Pulmonary arterial hypertension
- ii. Severe LV dysfunction (LVEF < 30%)
- iii. Previous PPCM (peri partum cardiomyopathy) with residual LV dysfunction
- iv. Severe left heart obstruction
- v. Marfan syndrome with aorta dilated to > 4cm

MCQ

1. Most common heart disease in developed world → Congenital
2. Most common heart disease in India → Rheumatic heart disease (RHD)
3. Most common RHD during pregnancy → Mitral stenosis

4. Most common congenital heart disease during pregnancy → ASD (Acyanotic)
5. Most common cyanotic heart disease during pregnancy → Tetralogy of fallot (TOF)
6. Heart disease with highest mortality in pregnancy
→ Eisenmenger syndrome/ pulmonary hypertension
7. Mortality rate with Eisenmenger's during pregnancy
→ 50%
8. Most common time of heart failure in pregnancy →
post-partum > Labour > 32-34wks of pregnancy
(Cardiac output (CO) – maximum in post-partum period)

During pregnancy

- Stenotic lesions → Worsen
 - Regurgitation lesions → Improve (due to decreased peripheral resistance)
 - Procedure of choice for mitral stenosis → Balloon valvuloplasty
 - Best time for surgery → 14wks

Fetal/Neonatal effects

00:20:21

- i. Abortion
- ii. IUGR
- iii. Prematurity
- iv. Congenital fetal heart disease

MCQS

- Q. Preferred mode of delivery → Vaginal
- Q. Can labor be induced → Yes
- Q. Choice of Analgesia → Epidural
- Q. Choice of Analgesia for pulmonary Hypertension/Aortic Stenosis → General Anesthesia
- Q. Second stage of labor → Cut short Ventouse > Forceps
- Q. Which drug to be avoided in post-partum → IV Ergometrine (Ergot alkaloid/ sympathomimetic)
- Q. To promote diuresis → give IV furosemide
- Q. Active management of 3rd stage of labor → Should be followed

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Indications for LSCS in heart disease 00:26:11

- i. Dilated aortic root > 4cm or aortic aneurysm
- ii. Severe acute congestive cardiac failure (CCF)
- iii. Recent myocardial infarct (MI)
- iv. Severe symptomatic Aortic stenosis
- v. Warfarin administration within 2 weeks of delivery
- vi. Need for emergency valve replacement

Indications for IE (Infective endocarditis)

prophylaxis

00:29:01

- i. Women with prosthetic valve/Prosthetic material in valve repair

- ii. Prior h/o of infective endocarditis
 - iii. Unrepaired cyanotic heart disease (or) Repaired lesion with residual defect
 - iv. Valvulopathy after heart transplant
- Q. When to administer IE prophylaxis → 30-60min before anticipated delivery time

Drug of choice for IE prophylaxis

- Ampicillin 2g IV
- Cefazolin/Ceftriaxone 1gm IV
- If Penicillin allergy – Clindamycin 600mg IV
- If Pt wants oral therapy → Oral Amoxycillin 2gm

Indications for Anti Coagulations

00:33:19

- i. Mechanical valves
- ii. Persistent atrial fibrillation
- iii. Left atrial thrombus
- iv. Past h/o of embolism

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Drug of choice for Anti coagulation:

- LMWH or UFH (1st choice since readily reversible)
- Unfractionated heparin (UFH) -Twice daily
 - Discontinue just before delivery
 - On the day of induction → Skip the dose

How to monitor

- APTT – should be twice the control
- Factor Xa levels → 0.35 to 0.7 u/mL

When to restart heparin

- 6hrs after vaginal delivery
 - 12hrs after C. section
- } Continue for 6wks

Warfarin

- If required can be given in 2nd & 3rd trimester



Continue till 36wks



Shift to heparin

Peri partum Cardiomyopathy

00:41:40

- Idiopathic dilated cardiomyopathy
- Genetic predisposition – familial/sporadic

Diagnostic criteria

1. Development of cardiac failure in the last month of pregnancy or within 5 months after delivery.
2. Absence of identifiable cause for cardiac failure
3. Absence of recognizable heart disease prior to last month of pregnancy
4. LV systolic dysfunction – EF < 45%
 - LV End Diastolic dimension → > 2.7 cm/m²

Proposed causes

00:46:10

1. Viral myocarditis - Parvovirus
 - Herpes virus
 - Epstein barr virus (EBV)
 - Cytomegalovirus (CMV)
2. Malnutrition
3. Inflammation & oxidative stress
4. Immune intolerance
5. Prolactin cleavage molecule (16 KDA prolactin)
6. Preeclampsia
7. Twin pregnancy
8. Smoking

Diagnosis

- 50% - Recovers in 6 months
- 30% - Recurrence rate

Contraception in heart disease

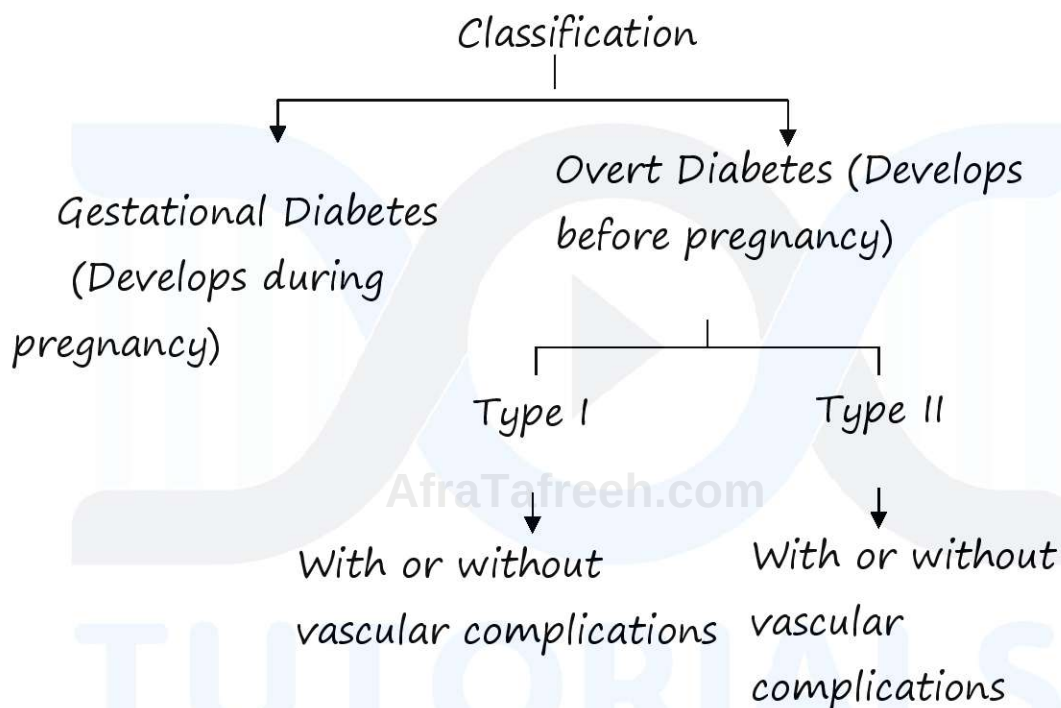
00:49:46

- Temporary: Barrier method
- Permanent sterilization
 - Do Vasectomy if heart is not well compensated
 - In Well compensated heart disease → Mini Laparotomy and tubectomy after 6 weeks

DIABETES COMPLICATING PREGNANCY

- i. Pregnancy → Predisposing factor
- ii. In pregnancy → Insulin sensitivity - ↓70% (Develops Insulin resistance)

Classification



Overt diabetes

00:01:05

- i. Affects Organogenesis – leads to anomalies
- ii. Require high doses of insulin for sugar control

Q. Overt diabetes is more dangerous than gestational diabetes

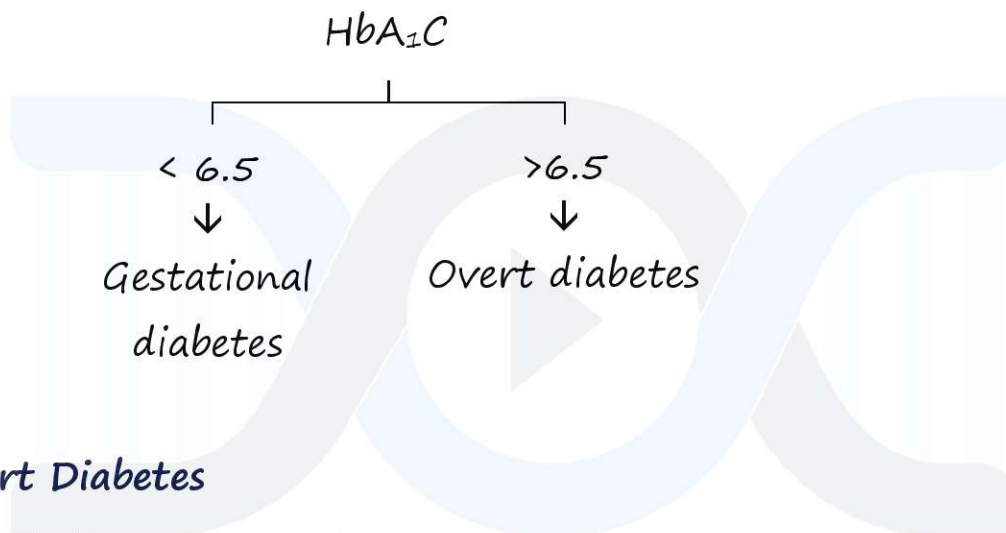
- ↑ Anomalies
- ↑ Intra uterine death

- $\uparrow$ IUGR
- $\uparrow$ Complication

Gestational Diabetes

00:05:06

- Any degree of glucose intolerance diagnosed for first time during pregnancy irrespective of gestational age & parity



Overt Diabetes

- Fasting $\geq 126\text{mg\%}$
- Random sugar $\geq 200\text{mg\%}$
- Hb A₁C > 6.5

LOGIN FOR SUCCESS

Guidelines for screening

00:09:26

ACOG Guidelines

- Incidence of diabetes less in developed countries
- They recommend 2 step tests
- Screening test -----> Diagnostic test

OGCT	OGTT
Oral Glucose Challenge test	Oral Glucose Tolerance Test
No fasting sample	Fasting sample required
1 value	Fasting, 1hr, 2hr, 3hr
Screening test	Confirmatory test

50g OGCT ('O' Sullivan Test)

00:13:06

- Done in High-risk women → Obesity

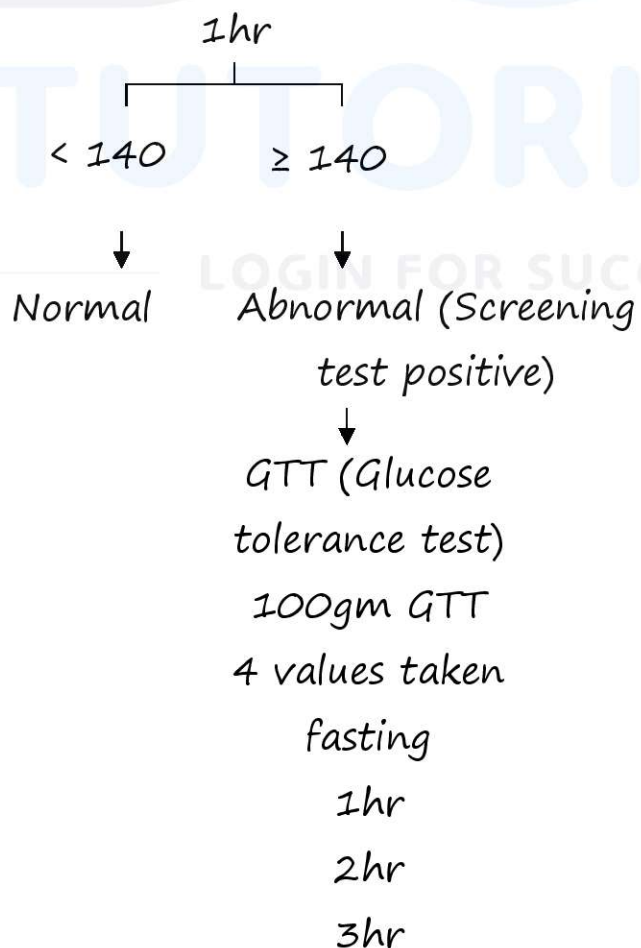
h/o of DM family

h/o DM,

Past h/o of diabetes

Anomalies baby

- 2 step techniques



100g GTT

	Carpenter & Coustan criteria	National Diabetes Data Group
		(NDDG)
Fasting	95	<105
1hr	180	<190
2hr	155	<165
3hr	140	<145

- If any two values exceed cut off → Diagnose DM

	G.D.M	Overt D.M
Fasting	95 - 125	≥126
1 hr	>180 - <200	≥200
2hr	>155 - <200	≥200
3hr	>140 - <200	≥200
HBA _{1C}	<6.5	>6.5

IADPSG

00:25:55

- International association of diabetes in pregnancy study group
- Followed in India

Criteria

- Universal testing
- Ideally done in 1st trimester at booking visit
- Direct diagnostic test → GTT
- 75gm GTT - 3 values

	G.D.M
Fasting	92
1hr	180
2hr	153

- Criteria – Any 1 abnormal value –Diagnosis Diabetes

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Priscilla white classification

Class	Onset	Fasting	2hr PP	Therapy
A ₁	Gestational	<105	<120	Diet
A ₂	Gestational	≥ 105	≥ 120	Insulin
	Age of onset	Duration	Vascular disease	Therapy
B	Over 20	< 10yr	None	Insulin
C	10-19	10-19	None	Insulin
D	Before 10	>20	Benign retinopathy	Insulin
F	Any	Any	Nephropathy	Insulin

R	Any	Any	Proliferative retinopathy	Insulin
H	Any	Any	Heart disease	Insulin

Complications

00:08:35

In 1 st Trimester	2 nd /3 rd Trimester
- Abortion (d/t glucose kinase mutation)-25%	- Macrosomia
- Anomalies – 11%	- Placentomegaly
- Acidosis	- Preterm
- Aneuploidies	- PROM
	- IUGR
	- Pre-eclampsia
	- Still birth
	- IUD
	- Polyhydramnios
	- Abruptio
	- Placenta previa

Macrosomia

Hyperglycemia (Mother)



By Facilitated transport
glucose crosses placenta

Hyperglycemia in fetus



Hyperinsulinemia

Increased Insulin like growth factor (IGF)



Macrosomia/Placentomegaly



↑ HCG Levels



↑ Chances of pre-eclampsia

IUGR

In Overt DM



Vasculopathy involving uterine artery



Blood supply to fetus ↓



causes IUGR

IUD/Still birth-causes

- Hyperglycemia
- Aberration in O₂ transport
- Constant fetal hypoxia
- During labour fetus needs more O₂ but in hypoxia -
→ Fetus develops asphyxia

MCQ's

- Q. Incidence of spontaneous abortions in overt DM - 25%
- Q. Incidence of Anomalies in overt DM - 11%
- Q. Most common complication in 2nd/3rd trimester
→ Macrosomia
- Q. Incidence of Macrosomia → 45%
- Q. Overall MC complication → Macrosomia
- Q. Incidence of preterm → 28%
- Q. Incidence of pre-eclampsia → 28%

Complications during Labour

00:22:27

- Shoulder dystocia
- Obstructed labour
- Postpartum hemorrhage
- Increases risk of C-section
- Increases risk of instrumental delivery

Complication in Post-partum

- Sepsis
- DVT
- Sub involution
- Lactation failure

Neonatal complications with diabetic pregnancy

00:25:17

1. Hyperinsulinemia
2. Hypoglycemia
3. Hypokalemia
4. Hypocalcemia
5. Hypomagnesemia
6. Polycythemia
7. Hyper bilirubinemia (Neonatal Jaundice)
8. Neonatal sepsis
9. Necrotising entero colitis
10. Respiratory distress syndrome
11. Cardiomyopathy (IVS -Inter ventricular septum)

Long term complications

- Impaired cognition
- Impaired language development
- ↓ IQ
- Poor memory
- Autism/Developmental delay

- Diabetes in future
- 3-5% Risk of type I DM
- 40% Risk of type II DM

Anomalies

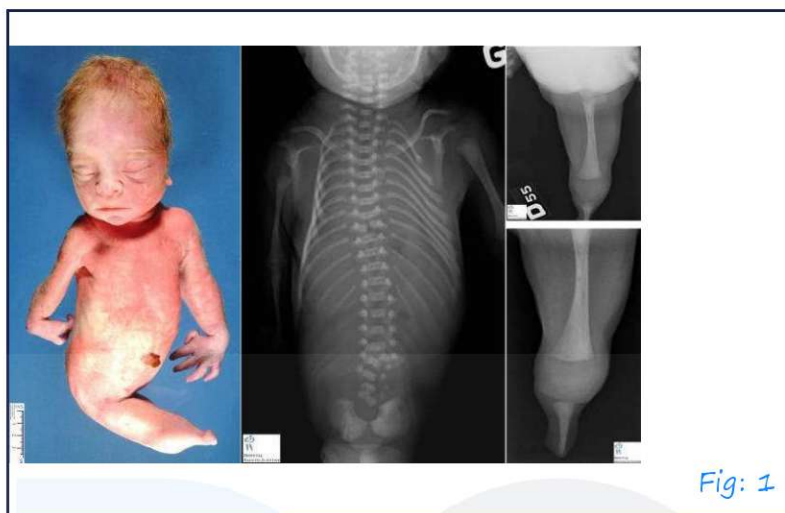
00:31:11

- Overall Incidence of anomalies in DM → 11%

HBA1C	Incidence of Anomaly
<6	2.5%
6-7	5%
>10	20% (advice termination of pregnancy)

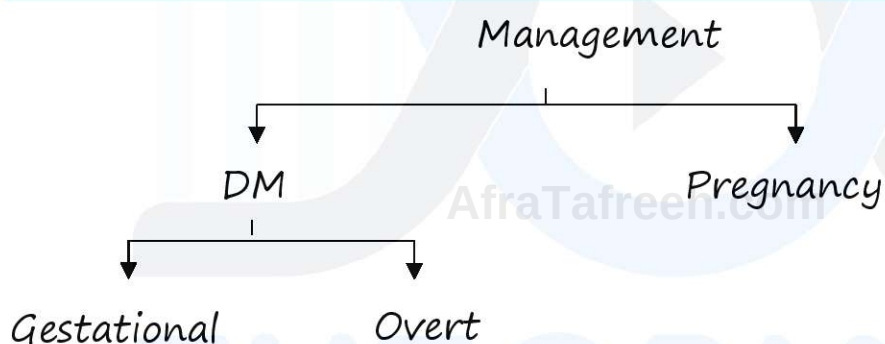
- MC system involved – Cardiovascular system > 50%
 - 5-fold Increased risk of developing Cardio vascular anomalies
 - 2nd MC system involved → Musculoskeletal system → Neural tube defect
 - Urogenital system → Renal agenesis, hydrouretero nephrosis, ureteric duplication
 - GIT → Duodenal atresia, Imperforate anus
 - M/c cardiovascular abnormality → VSD
 - Most specific cardiovascular abnormality → TGA (Transposition of great arteries)

- Sacral agenesis -also known as *caudal regression syndrome* -Most specific for diabetes^Q

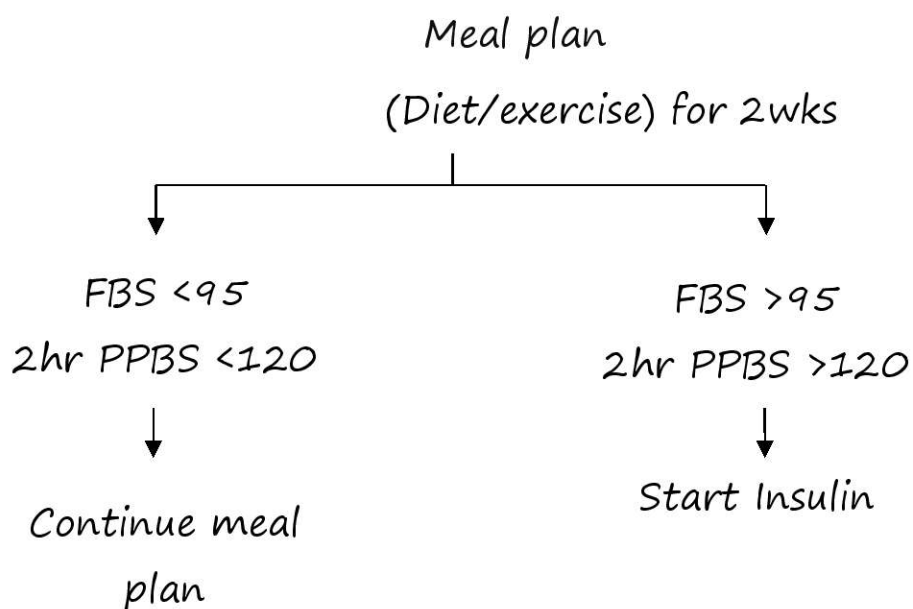


Management

00:42:23



- Gestational diabetes mellitus (GDM) management



Overt DM

- Meal plan + Insulin

Calorie

- Normal BMI $\rightarrow$ 30-35 Kcal/K/d
- BMI $>$ 30 $\rightarrow$ 25 Kcal/Kg/d

Diet – GDM	Overt
Carbohydrate – 40%	55%
Protein – 20%	20%
Fat – 40%	25%

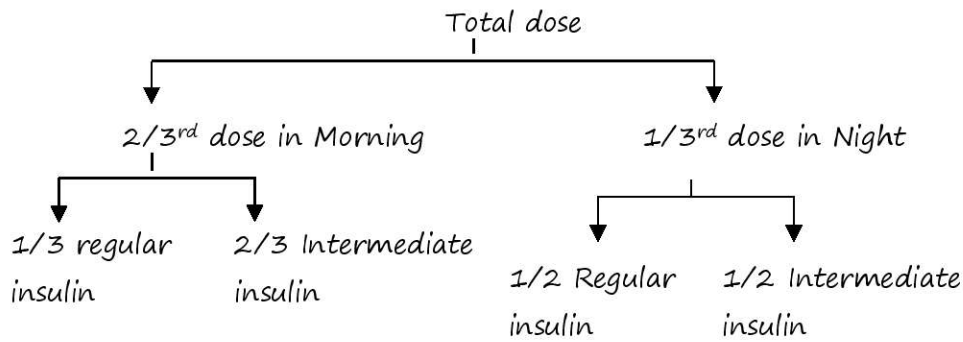
OHA (Oral Hypoglycemic Agent)

- OHA $\rightarrow$ Cross placenta, Teratogenic (except $\rightarrow$ Glyburide & metformin) Glyburide $\rightarrow$ Cross placenta $\rightarrow$ Cause Respiratory distress/Neonatal hypoglycemia Not used much

OHAs are not as effective as insulin in controlling DM.

Insulin

- Rx of Choice
 - 0.7 to 1 unit/kg/day in divided doses
 - Intermediate + short acting



Delivery

00:53:37

- Women on meal plan → Deliver @ 40wks
- Women on Insulin → 39wks
- Preferred mode of delivery → Vaginal delivery
- Macrosomia → Fetal weight → >4.5kg → LSCS
- Q If planning for elective LSCS <39wks → Give full course of steroids

Insulin during labour

- Hourly CBG (capillary blood glucose) and normal saline infusion
- If blood sugar < 70mg% → Start 5% Dextrose
- If blood sugar > 100mg % → Start Insulin Infusion (@1.25u/hr)
- If planning to induce, skip the morning dose of insulin and try to maintain sugar between 70-100.

THYROID DISORDERS IN PREGNANCY

Hyperthyroidism in pregnancy

- Incidence: 2 to 12/1000 pregnancies
- Causes:
 - Hashimoto's thyroiditis → M/C cause due to Anti TPO Antibodies
 - I₂ deficiency
 - Post ablation
 - Treatment for graves' disease (Over-correction)

Adverse effects on pregnancy

- Infertility
- Abortion
- Preterm labour
- Preeclampsia
- Diabetes
- Abruption
- Impaired psychomotor development
- ↓ School performance
- ↓ IQ
- Daily I₂ requirement during pregnancy → 220 mcg/day
- Daily I₂ requirement during lactation → 290 mcg/day

Treatment

- Levothyroxine 1 -2mcg/kg/day → 100mcg/day

Post-partum thyroiditis

00:05:22

- Inflammation of thyroid gland during post-partum period
- Suddenly develops hypothyroid symptoms in post-partum period
- Associated with
 - Anti-TPO antibodies
 - Post-partum depression
- Incidence → 5-10% among postnatal women with in 1yr after delivery
- 1st phase
 - Destruction induced thyrotoxicosis
- Features are:
 - Abrupt onset
 - Small Painless goitre
 - Fatigue
 - Palpitation (Respond to β blockers)
- 2nd phase
 - Hypothyroid phase
 - Thyromegaly → after 4 to 8 months of onset of disease

Thyrotoxicosis in pregnancy

00:10:56

- **Indicence:** 2 to 17/1000 pregnancies
- **Causes** → most common cause of hyperthyroidism → **Grave's disease** Due to Antibodies Stimulating TSH receptor

Features:

- Thyromegaly
- Tachycardia
- Exophthalmos
- Failure to gain weight
- Markedly depressed TSH levels

Pregnancy outcome

- Preeclampsia
- Heart failure
- Abortion
- Preterm delivery
- Still birth
- IUGR
- Fetal hypothyroid/hyperthyroid
- Fetal Goitre
- Hydrops (Non-Immune hydrops)
- Hearing loss

Fetal Neonatal effects

- Fetal thyrotoxicosis → d/t placental transfer of antibodies stimulating TSH-Receptors
- Hypothyroidism
 - i. Goitre → d/t placental transfer of Antithyroid drug
 - ii. Non – goitrous → due to placental transfer of TSH – Receptor Blocking Ab

Management of thyrotoxicosis

00:17:29

- DOC: Propyl thiouracil (50-150mg TID)
 - Unsafe drugs
 - i. Methimazole → Esophageal atresia, choanal atresia, aplasia cutis (May 2020 AIIMS)
 - ii. Carbimazole

Propyl thiouracil

- Hepatotoxicity
- Transient leucopenia
- ANCA – Vasculitis

Surgery

- If No response to medical treatment Subtotal thyroidectomy should be Done in 2nd trimester

Radioactive iodine ablation

00:21:42

- C/I during pregnancy
- Unintentional exposure → termination of pregnancy
- Postpone pregnancy – for 6 months after exposure



HYPERTENSIVE DISORDERS IN PREGNANCY – I

(DEFINITION AND RISK FACTORS)

Hypertension in Pregnancy

00:00:30

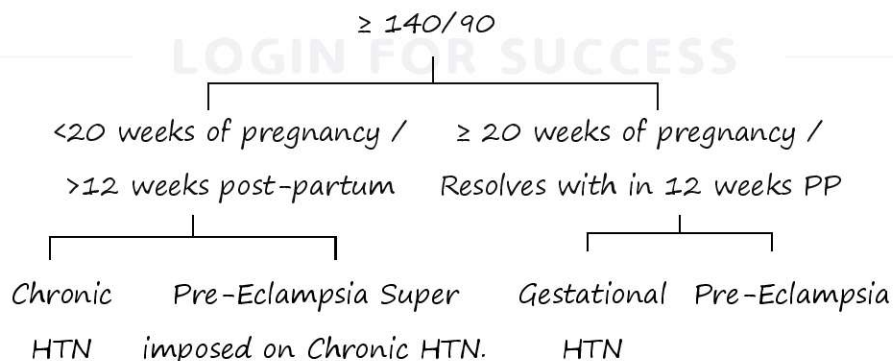
- Incidence of HTN in pregnancy → 5 - 10%
- Incidence of preeclampsia → 4-5%

In India most common of maternal mortality
↓
Post-partum Hemorrhage

Definition:

- Any women with blood pressure $\geq 140 / 90$ mmHg are considered hypertensive

Korotkoff sound – V
↓
Diastolic Blood pressure



Preeclampsia

00:05:56

- Hypertension
- Proteinuria $\geq 300\text{mg} / 24$ hours /
1 + Dipstick /

Urine protein creatinine ratio ≥ 0.3

(or)

- Any of the following complications
 - Thrombocytopenia – (Platelet $< 1,00,000$)
 - Renal Insufficiency $\rightarrow$ (Sr. Creatinine > 1.1 mg%)
and Doubling of base line S. Creatinine value
 - Liver involvement – S. Transaminase levels twine –
normal
 - Cerebral symptoms – Headache, visual disturbances
 - Pulmonary edema

Trace – 0.1 g/L

1 + – 0.3 g/L

2 + – 1 g/L

3 + – 3 g/L

4 + 10 g/L

Imminent symptoms / Impending symptoms /
warning symptoms

00:14:51

1. Headache (Occipital headache)
2. Blurring of vision – Scotoma
3. Diminished urine output
(Urine O/P < 100 ml for 4 hours)
4. Epigastric pain / Rt upper Quadrant pain

Epigastric / Rt upper quadrant pain
↓
Due to Hepatocellular Necrosis /
Stretching of Glisson's capsule

	<i>Non- Severe</i>	<i>Severe</i>
Time Of Delivery	37 weeks	34 / < weeks
Proteinuria	+/-	+/-
Headache Usual Disturbance	-	+
oliguria		
Convulsion	-	+ (Eclampsia)
S. Creatinine	-	Elevated
Thrombocytopenia	-	+
Elevated Transaminase	mild increase	↑↑
Pulmonary Edema	-	+
IUGR	-	+

Risk factors

00:24:54

1. Extremes of age – young and women > 35 years
2. Nulliparous
3. Primiparity
4. African / American
5. Metabolic syndrome
6. Hyperhomocystinemia
7. Male fetus

8. HIV positive
9. Sleep disorders
10. Past h/o preeclampsia – most common
11. Chronic hypertension – second most common
12. Diabetes
13. Multiple pregnancy
14. Obesity
15. APLA / SLE

Other Risk Factors

1. Previous h/o abruption / still birth
2. Chronic kidney disease
3. ART
4. Rh incompatibility
5. Trisomy 13

TUTORIALS

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HYPERTENSIVE DISORDERS IN PREGNANCY – II

(PATHOPHYSIOLOGY AND MARKERS)

Etiology

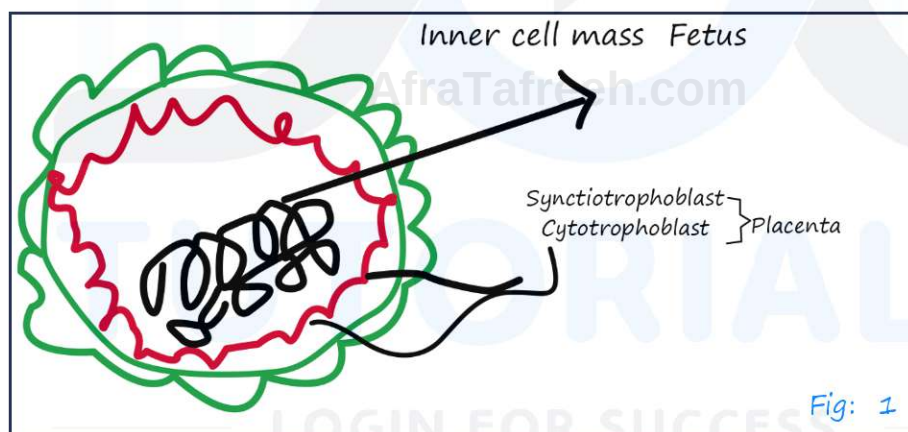
00:00:15

1. Abnormal trophoblastic invasion
2. Immunological factors
3. Maternal maladaptation
4. Genetic factors

1. Abnormal Trophoblastic Invasion

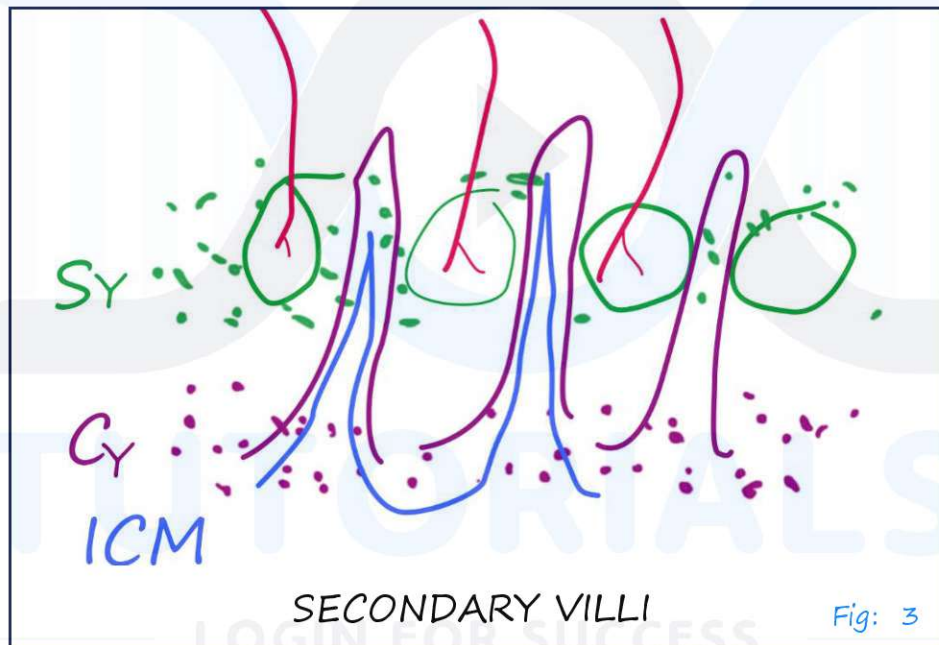
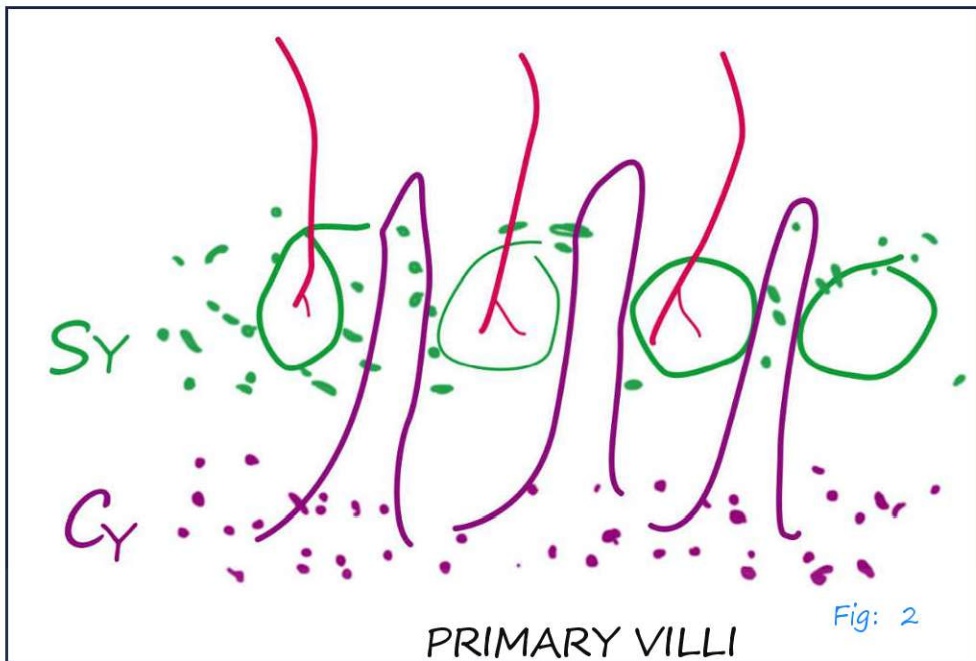
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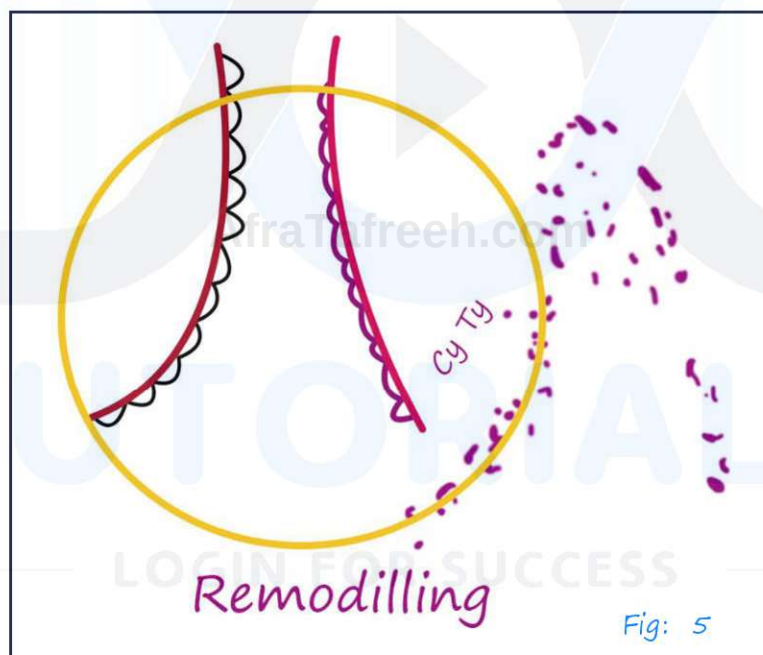
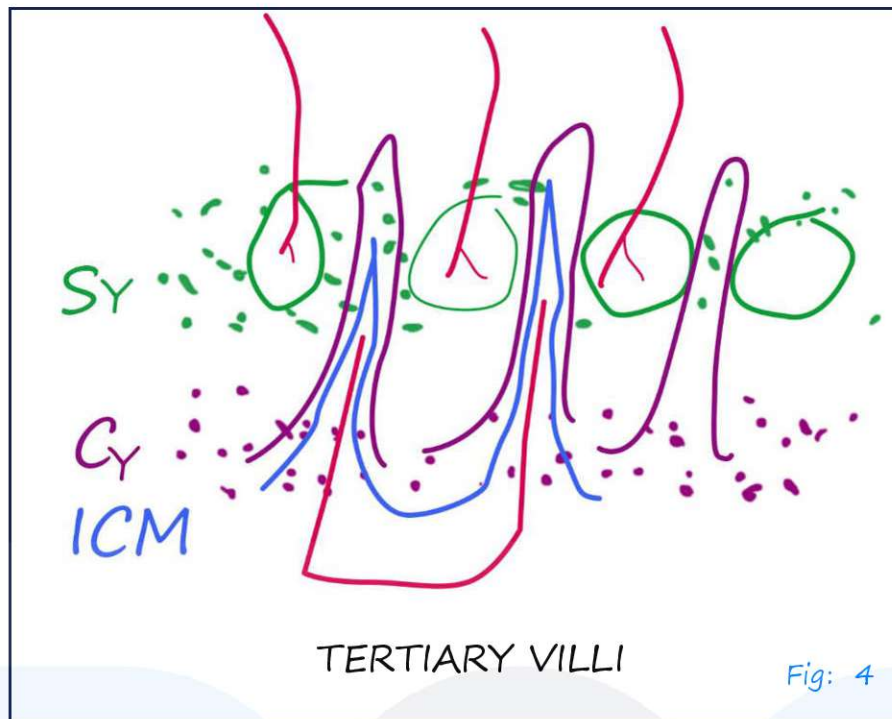
Normal Trophoblastic invasion



Cut on section

- Decidua basalis
- Syncytiotrophoblast
- Cytotrophoblast
- Inner cell mass





Normal Invasion:

Spiral arteries (which supply decidua)

↓ penetrate

Lacuna (In syncytiotrophoblast)

↓

In between Lacunae, Cytotrophoblastic projections come

↓

Primary villi formed (Syncytiotrophoblast is invaded by
cytotrophoblast)



Cytotrophoblast cells – replace original endothelial cells
of spiral arteries



Maternal spiral vessel



Low – resistance vessel.

If it does not happen



Vessel does not become low resistance vessel (high
resistance)



No decrease in Diastolic blood pressure (DBP)



Increase DBP



Pre – eclampsia.

2. Immunological factors

00:09:18

- Normal pregnancy



Mother develops blocking abnormal against foreign Ag.
(Fetus)



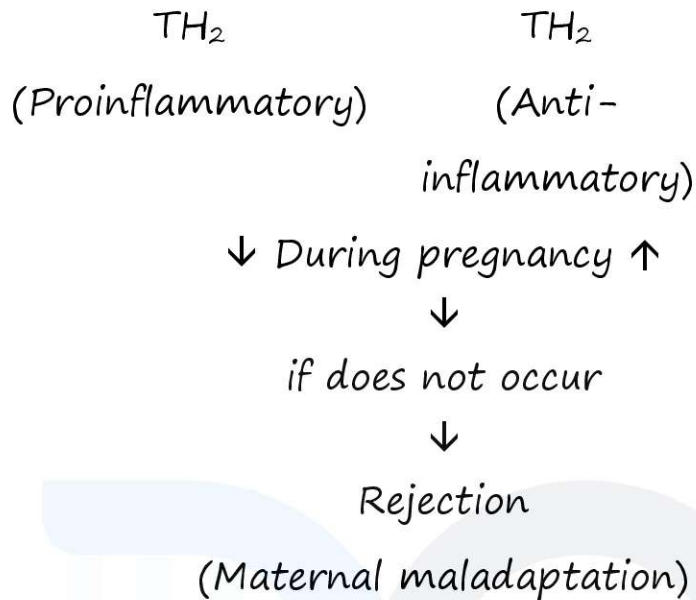
If mother does not develop Abnormal



Defective maternal immune tolerance

3. Maternal maladaptation

00:11:40



4. Genetic factors

00:13:10

1. MTHFR gene (Methyl Tetrahydrofolate reductase gene)
 2. Factor V Leiden gene
 3. HLA gene
 4. NO synthase III gene
 5. Angiotensinogen gene
 6. ACE gene (Angiotensin converting enzyme)
 7. Lipoprotein lipase.
- Oxidative stress
 - ↓
 - Reactive oxygen species
 - ↓
 - Vessel wall injury
 - ↓
 - Vasospasm

Markers in Pre-eclampsia

- Increase
 1. Theca lutein cyst
 2. HCG
 3. Growth hormone
 4. Soluble FMS like tyrosine kinase
 5. Leptin
 6. Endothelin
 7. Soluble
 8. Cardiac troponin
 9. D-Dimer
 10. Increase consumption of factor V111
 11. Fibrinopeptides A, B
 12. Increase ANP, BNP
- Decrease
 1. Decrease PGI_2 , $7xA_2$
 2. Decrease refractoriness to AT X
 3. Decrease Nitric oxide
 4. Decrease VEGF
 5. Decrease PlGF

Hyperuricemia
Hypocalciuria

} Markers of preeclampsia

HYPERTENSIVE DISORDERS IN PREGNANCY

(END ORGAN CHANGES PREDICTION AND PREVENTION)

End organ changes

00:00:01

- Kidney

Most commonly Involved organ

- Decrease renal perfusion
- Decrease GFR (Glomerular Filtration Rate)
- Glomerular endotheliosis – characteristic lesion seen in preeclampsia
- Hyperuricemia
- Hypocalciuria
 - Low calcium reserve in the body is a predisposing factor for pre-eclampsia
 - Pre – eclampsia can be predicted by low calcium excretion in urine
- Acute tubular necrosis
- Increase urine sodium concentration
- Increase urine osmolality
- Increase plasma – creatinine ratio

Liver

00:03:40

- Periportal hemorrhage
- Sub – capsular hematoma

Brain

00:04:20

- PRES – Posterior – Reversible Encephalopathy Syndrome
 - Due to cerebral hyper perfusion
 - Present with → Headache, field defects
- Cortical and sub cortical petechial hemorrhage: presents with headache and scotoma

Predictors

00:06:40

1. Roll over test

- Measure Blood Pressure in left lateral position, then turn to supine position and measure Blood Pressure (if difference in DBP is <20 mm Hg → normal if difference in DBP is >20 mm Hg → predicts pre-eclampsia)

2. Angiotensin sensitivity test

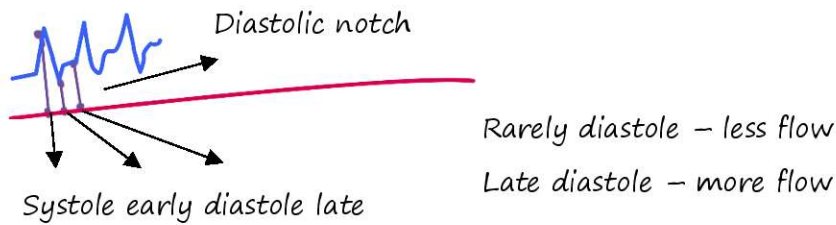
- Measures Blood Pressure before and after giving angiotensin, if difference in DBP is > 20 mm Hg → risk for preeclampsia

3. Isometric exercise test –

- Measure blood pressure before and after pressing the smiley ball with the hand – , if difference in DBP is > 20 mm Hg → risk for preeclampsia

4. Uterine artery doppler – done at 24 weeks

- In 1st trimester:



Early and late diastole variation is seen only in 1st trimester when 2nd wave trophoblastic invasion has not yet occurred

- In 2nd trimester:
 - 2nd wave trophoblastic invasion occurs, due to which the spiral arteries penetrate deep into the lacuna



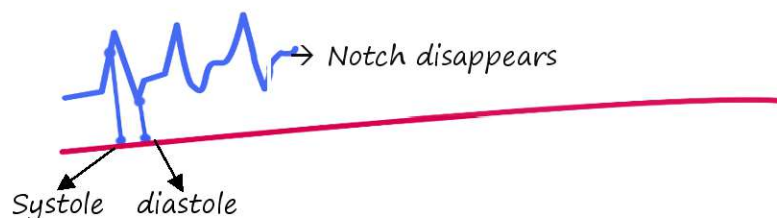
More segment of spiral arteries relaxes



Pressure and resistance – drops



Blood flow improves



- Persistence of early diastolic notch is a predictor of preeclampsia.
- In present day early diastolic notch is a better predictor for IUGR only than for preeclampsia

5. Substances in serum like HCG, PAPP-A, Inhibin, Activin, Procalcitonin, kisspeptin, CRH, were studied but were not useful

6. Uric acid

- Hyperuricemia – predictor of cellular turnover.
- More cellular turnover / more hypoxia in the placenta is marked by high levels of hyperuricemia

7. Microalbuminuria

8. Fetal fibronectin

9. Cell free fetal DNA / maternal serum free fetal DNA

10. Ratio of soluble FMS like Tyrosine kinase and PlGF.

1,2,3,6,7,10 – are the only useful predictors of pre-eclampsia

Prevention

00:18:30

- Useful in preventing Eclampsia
 - Regular exercise
 - Smoking
 - Bed rest
 - Statins
 - Metformin
 - Aspirin and Heparin – promising for APLA
- Not useful in preventing preeclampsia

- Low – salt diet
- Calcium supplements
- Fish oil
- Diuretics
- Anti hypertensives (Not prophylactic use)
- Antioxidants.



PREGNANCY AFTER PREVIOUS CAESAREAN

SECTION

Birth after previous cesarean birth

- Also called as post cesarean pregnancy

Effects and previous cesarean scar on pregnancy and labour:

- Abortion
- Preterm labour
- Increase operative interference (repeat C - section, forceps, vacuum)
- Placenta previa
- Placenta accretes
- Post-partum hemorrhage
- Peripartum hysterectomy

Incidence of p. previa after

1C - section → 1%^Q

2C - section → 1.7%

3C - section → 2.8%

^QIncidence of P. Accrete is 11-14 percent after 1-c-section

Effect of pregnancy on scar

00:05:26

Scar ruptures → classical c-section scar > lower segment.

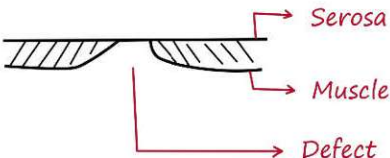
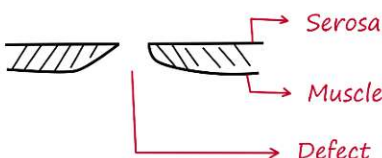
Scar rupture	timing
Classical c-section scar	– 4-9% – during late pregnancy/labour
Lower segment scar	– 0.5% – during labour.

Scar dehiscence:

Asymptomatic thinning of uterine scar without involving peritoneal coat without hemorrhage.

Difference between uterine dehiscence and uterine rupture

00:09:51

Uterine dehiscence	Uterine rupture
Disruption of uterine muscle with intact uterine serosa  Serosa Muscle Defect	Disruption of uterine muscle extending to involve the serosa or extension to bladder or broad ligament  Serosa Muscle Defect

Difference between lower segment and classical scar

00:63:20

Lower segment scar	Classical scar
Lower segment scar 	Classical scar 
Thin margins – perfect oppositions No pockets – formation	Thin margin – difficult to appear Pockets are formed filled with – blood and fibrin -gutter formation
Remains inert/intact while healing	Upper segment ↓ Contracts and retracts ↓ Sutures become loose ↓ Imperfect healing
Scar stretches along the line of scar during future pregnancy	Scar stretches at right angle ↓ Maker under able to rupture

Placement attachment is unlikely	Placement attachment is likely ↓ Trophoblastic penetrating or herniation of amniotic sac through gutter
Rupture is rare, If accesses, during labour	Scar-week Rupture during lab pregnancy.
Incidence of rupture → 0.5%	→ 4-9%

Management of previous C- section pregnancy

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00:21:38

i. Planned vaginal birth after caesarean (VBAC)

ii. Elective repeat c section.

TOLAC: - Trial of labor after c. section.

LOGIN FOR SUCCESS

Contraindications for VBAC: QQ

- Previous uterine rupture (Recurrence rate- 5%)
- Previous classical C section
- Uterine rupture with hysteroscopic septal resection
- Myomectomy with breach into cavity
- Other obstetric clinical for vaginal delivery like placenta Previa, major CPD major.

Special circumstance where VBAC can be performed:

- Women with two or more prior LSCS
- Post dated pregnancy
- Twin gestation
- Fetal macrosomia
- Antepartum still birth/IUD
- Maternal age > 40
- Preterm labour

VBAC can be done with – caution

Factors increasing the likelihood of successful VBAC

00:35:06

1. Previous vaginal delivery
2. Previous VBAC^Q
3. Greater maternal height
4. Maternal age <40
5. BMI <30
6. Gestational age <40 weeks
7. Spontaneous onset of labour
8. Vertex presentation
9. Engaged head/favorable station
10. High BISHOP'S SCORE
11. White ethnicity

^QSingle best predictor of successful VBAC → past h/o
successful VBAC (or) vaginal delivery

Factors increasing risk of rupture:

- Short inter delivery interval
- Postdated pregnancy (>40 weeks)
- Maternal age >40 year
- Obesity
- Low bishop's score
- Macrosomia
- Thin lower segment in USG (<2mm)
- Need for induction/augmentation of labour

Success rate of VBAC After, previous c-section → 72-75%

Success rate of VBAC in women with previous vaginal delivery → 85-90%.

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Factors of previous caesarean affecting VBAC

success

00:45:40

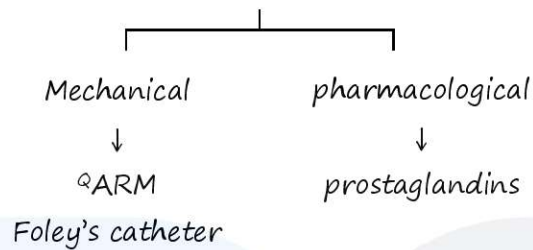
- Increase success
 - Previous C – section for malpresentation (non-recurring indication)
 - Previous C – section for failed instrumental delivery
- Decrease success
 - Previous C – section for dystocia
 - Previous C – section for fetal distress
 - Previous emergency C – section
 - Previous failed induction.

Induction/augmentation

can be done at 41 weeks, but increase of C – section/rupture.

i. Induction

Can be done by 2 methods



ii. Augmentation



Done by oxytocin

But increases Risk of rupture when given at
>20MJU/min

Ideal technique of induction → Mechanical foley's catheter.

Intrapartum management:

- VBAC is conducted in place where facilities for C – section Neonatal resuscitation are available
- Continuous CTG/Electronic fetal HR monitoring
- Epidural analgesia → can be given.
(But increase need of analgesia – sign of impending rupture)

QQ Classical features associated with scar Rupture

00:56:30

1. Abnormal CTG → Most consistent finding of impending scar rupture
 2. Severe abdominal pain → in between contraction
 3. Acute onset scar tenderness
 4. Abnormal vaginal bleeding
 5. Hematuria
 6. Cessation of contractions
 7. Tachycardia, hypotension, fainting, shock
 8. Loss of station/recording of presenting part.
 9. Change in abdominal contour
 10. Difficulty in picking up fetal HR
- If women do not want to undergo VBAC

↓

Then go for Elective Repeat C – Section.

Elective repeat C – section: (ERCS)

When: only after 39 completed weeks

If deliver <39 weeks → give steroids for lung maturity

Antibiotics prior to skin incision – must

Thromboprophylaxis should be given

Extra edge

	VBAC	ERCS
Uterine rupture	0.5%	0.02%
Blood transfusion	2%	1%
Maternal mortality	4/1 lakh	13/1 lakh
Transient neonatal respiratory distress	2.3%	4.6%



INTRAHEPATIC CHOLESTASIS OF PREGNANCY

Classification of Jaundice

00:00:24

Obstetric causes	Non-Obstetric causes
- Acute fatty liver of pregnancy	- Cirrhosis
- Intrahepatic cholestasis	- Viral hepatitis
- Hyperemesis	- Varices
- Preeclampsia	

Intrahepatic Cholestasis of Pregnancy

00:02:05

- Intrahepatic cholestasis of pregnancy /Also known as Recurrent jaundice of pregnancy/Cholestasis hepatosis / Icterus gravidarum
- Common in multiple pregnancy
- Significant genetic influence present
- Incidence – 1 in 500 to /1 in 1000 pregnancies

Pathogenesis

00:04:32

- Mutation in **ABCB 4 gene / ABCB 11 gene**
- ABCB 4 encodes MDR protein 3
- ABCB 11 encodes a protein which is bile salt export pump.

- Other genes are – farnesoid x receptor, ATP B1 – encodes transporting ATPase
- **Azathioprine** – decreases canalicular transport of bile acid

Clinical features

00:07:38

- Pruritis – (itching) – more in soles & palms
- more commonly seen during night time, occurs in 2nd & 3rd trimester of pregnancy
- Jaundice (incidence – 10%)

Biochemical changes

00:09:10

- Bile acid > 10m mol/L
- Increased Bilirubin – 2–5 mg%
- Alkaline phosphatase – increased
- AST – Normal to 250 IU/L

Liver Biopsy

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00:10:52

- Shows cholestasis with bile plugs – more commonly seen in centrilobular region
- No inflammation
- No necrosis
- These changes disappear after delivery. And recur with subsequent pregnancy.

Complications

00:12:55

Maternal	Fetal
- Pre eclampsia	- Fetal distress
- GDM	- Prematurity - Respiratory distress syndrome
	- Neonatal morbidity/ mortality

Treatment

00:14:42

- Induction between 38-39 weeks
- For itching, drug of choice - UDCA - Urso deoxy cholic acid
- Antihistamines
- Topical emollients

TUTORIALS

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ACUTE FATTY LIVER OF PREGNANCY

- It is the most common cause of acute liver failure during pregnancy
- Accumulation of micro vesicular fat-liver spongy, swollen, yellow in colour
- Incidence 1 in 10,000 pregnancies

Etiopathogenesis

00:01:21

- Due to mitochondrial abnormalities of fatty acid oxidation
- Recessive inherited condition
- Similar to Reye's syndrome
- Children born from mother of acute fatty liver of pregnancy have high chances of developing Reye's syndrome
- Mutation in genes encoding LCHAD, MCAD, CPT-1
- LCHAD – long chain hydroxyl acyl CoA dehydrogenase
- MCAD – Medium chain hydroxyl acyl CoA dehydrogenase
- CPT-1 carnitine palmitoyl transferase
- AFLP → at high risk for preeclampsia & HELLP syndrome
- Intrahepatic cholestasis is also associated with preeclampsia & HELLP syndrome

Clinical Features

00:05:50

- Nausea/Vomiting **Main C/F**
- Abdominal pain
- Anorexia
- Epigastric pain
- Jaundice
- Seen in late in pregnancy [3rd trimester]
- Hypertension/proteinuria & edema seen in 50% patients

Pathophysiology

00:07:44

RBC membrane is devoid of cholesterol



Fragile RBC



Hemolysis – [No hemolysis in intra hepatic cholestasis]



Leukocytosis, thrombocytopenia



Increases LDH and Echinocytosis

Liver failure – hypocholesteremia



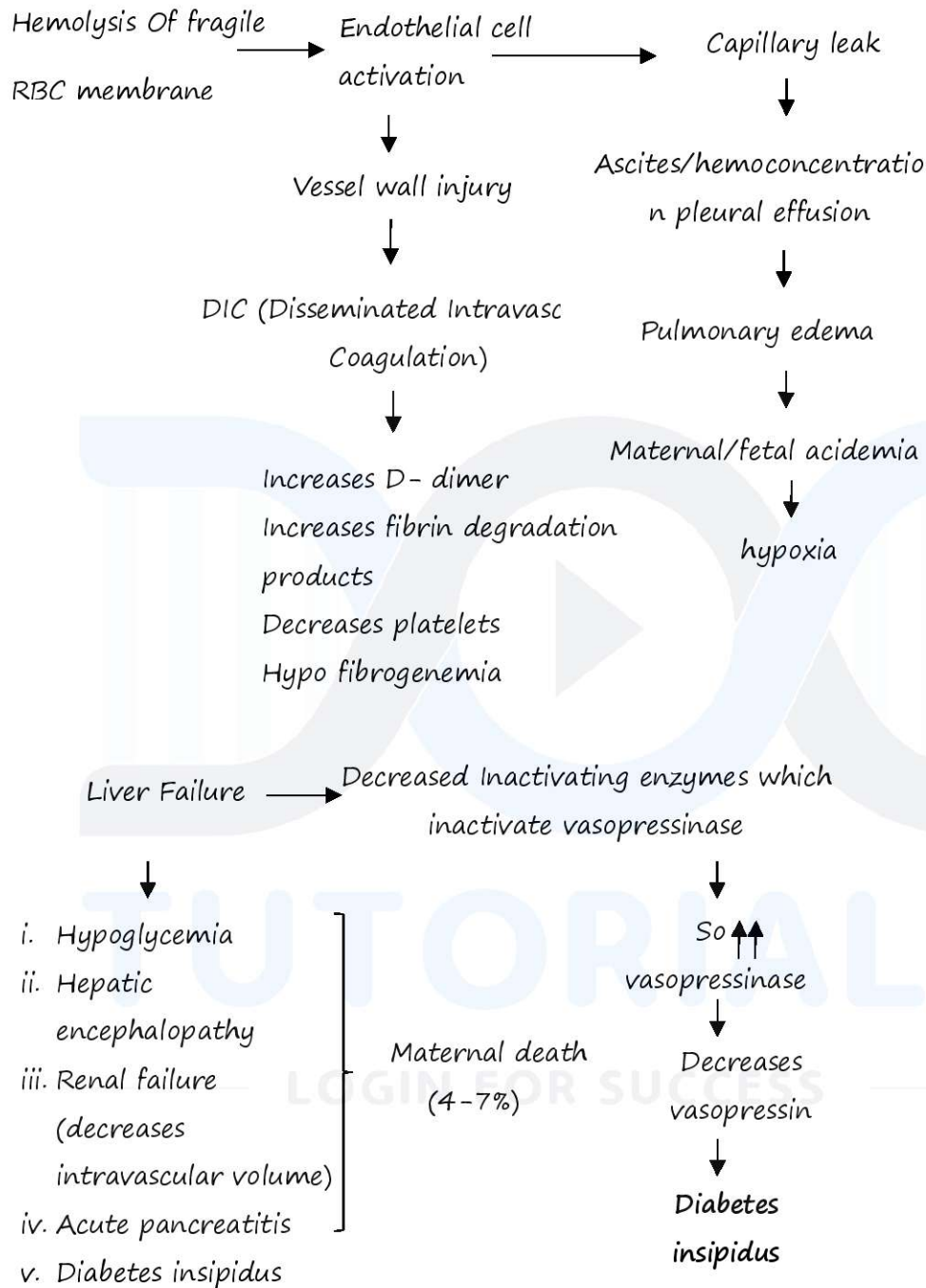
Hypoalbuminemia



Increases bilirubin – 4-10mg%

Increases AST – 200-800IU

Liver failure → decreases clotting factor → hypo
fibrinogenemia



Treatment

00:19:11

- Immediate termination of pregnancy
- If factors are favorable → Trial of labor
 - C-section – obstetric indication
- In DIC
 - Blood & blood product
 - Plasma exchange
- Liver transplant
- Recurrence
- is very rare.

Mortality rate – 4-7%



VIRAL HEPATITIS

- Hepatitis is one of the causes for jaundice during pregnancy but not the MC cause
- MC cause for acute liver cell failure is acute fatty liver of pregnancy
- There are 5 types of hepatitis viruses
 1. Hepatitis-A
 2. Hepatitis-B
 3. Hepatitis-C
 4. Hepatitis-D
 5. Hepatitis-E

Hepatitis-A

00:00:47

- Hepatitis-A has no parent to child transmission at all
- Not teratogenic (hepatitis-A)
- Hepatitis-A is not transmitted through breast milk
- Course of the disease is the same as the non-pregnant lady
 - Both Active and Passive vaccination are allowed during pregnancy

Hepatitis-B

00:03:23

- MC viral hepatitis during pregnancy but not most dangerous
- And has trans placental transmission

- So, it is important to screen hepatitis-B during pregnancy and if positive antivirals can be started
- 30-50% of HBV infection are due to vertical transmission
- Course of the disease is not altered by pregnancy or course of pregnancy not altered by disease
- Usually, asymptomatic
- Two modes of transmission
 - i. Trans placental
 - ii. Transmission during delivery is **MC**
- Screening for both hepatitis C & B done during pregnancy if positive for hepatitis-B → to prevent mother to child transmission immunization can be done
- Transmission rates not only depend on the hepatitis surface antigen but also on the infectivity (Surface ag-HbeAg)

	(HbsAg) Surface Ag	HbsAg with HbeAg
Without immuno prophylaxes	10-20%	90%
With immuno prophylaxis	-	10%

For immuno prophylaxis

- Combined active and passive immunization
- Neonate 0, 1, 6 (immediately after birth)
- Hepatitis-B vaccination allowed during pregnancy

For Mother who is HbsAg (positive)

1. Tenofovir (Doc)
2. Telbivudine → 2nd choice
3. Lamivudine: S/E rapid development of resistance and not effective in 3rd trimester
4. Breast feeding can be allowed

Hepatitis-C

00:13:14

- Not teratogenic
- Course of the disease is the same
- Transmission rate-Mother to child → 7% mainly during labor and peripartum period
- Mode of delivery both in hepatitis B & C does not affect much breast feeding is allowed

Antivirals

- | | | |
|---------------------------|---|-----------------------|
| - Alpha interferon | } | Not used in pregnancy |
| - Ribavirin (teratogenic) | | |

Hepatitis-E

00:15:36

- Course of the disease is much altered with pregnancy

- Most dangerous hepatitis during pregnancy – cause Acute fulminant liver failure/hepatitis
- No mother to child transmission; no vaccines available
- No approved vaccines

Disorder	Onset	C/F	AST	Bilirubin	Creatinine	HCT	Platelet	Fibrinogen	D-dimer	PT
Hyperemesis	1 st trimester	Nausea/vomiting	Normal to 200	Normal to 4	↑	↑↑↑	Normal	↑	normal	Normal
Cholestasis	Late	Itching	Normal to 200	Normal to 5	Normal	Normal	Normal	Normal	Normal	Normal
Fatty liver	3 rd trimester	Nausea/vomiting/HTN	200 - 800	4-10	↑↑↑	↑↑	↓	↓↓↓	↑	↑↑
Pre-eclampsia	2 nd or 3 rd trimester	HTN/Edema	Normal to 300	1-4	↑	↑	↓↓	Normal	↑	Normal/hemolysis present
Viral hepatitis	Any gestational age	Jaundice	200-1000+	5-20	Normal	↑	↓	Normal	Normal	↑ Hemolysis absent

LOGIN FOR SUCCESS

HIV IN PREGNANCY

HIV 1 > HIV 2

Testing

00:00:27

- Opt out approach
- If pregnant lady is not willing to get tested → don't force → consider positive & take precautions while delivering
- 1st screening at booking visit



Repeat testing at/ before 36 wks.' for all pregnant women.

Transmission rates

00:01:40

- Depends on viral load
- Transmission rate
 - >30,000 copies/ml – 23%
 - < 50 copies/ml – Nil
- Transmission with Invasive procedures
- Without ART – Risk doubles
- with ART – No ↑ in risk

Timing of transmission

00:03:45

- During pregnancy, < 36 weeks → 20%
- Last few weeks before delivery → 50% (highest)
- Intrapartum → 30%

- Breast feeding → 30-40%

Recommendations on ART

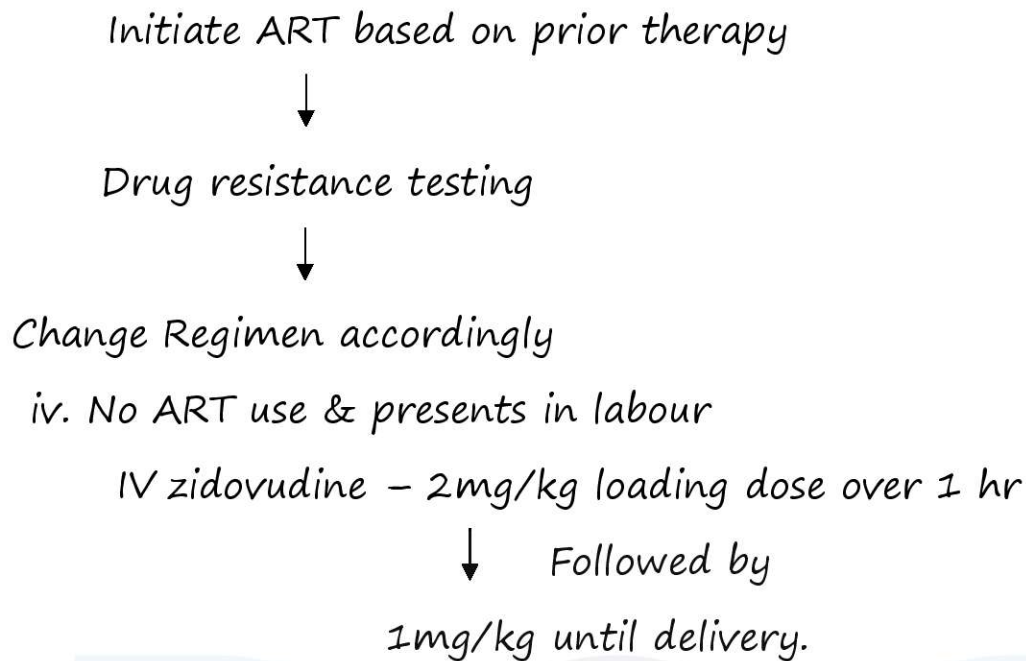
00:04:19

- All HIV positive pregnant women should be put on ART, continue lifelong irrespective of CD₄ count & viral load
- Atleast 3 drugs should be given → 2 NRTI + 1 PI
- Preferred Drugs
- NRTI (Nucleoside Reverse Transcriptase Inhibitor)
 1. Abacavir
 2. Lamivudine
 3. Tenofovir
 4. Emtricitabine
- PI – Protease Inhibitor – Atazanavir, Ritonavir
- II – Integrase Inhibitor – Raltegravir

4 Categories of Patients Seen Clinically

00:06:08

1. Taking ART & becomes pregnant
2. ART naïve (not used any medications)
3. Prior ART use but not currently
4. No ART use & presents in labour
 - i. Taking ART & becomes pregnant: Continue same treatment if response is good.
 - ii. ART naïve patient Initiate ART (as early as possible) (2NRTI + Ritonavir booster PI or Integrase inhibitor)
 - iii. Prior ART use but not currently



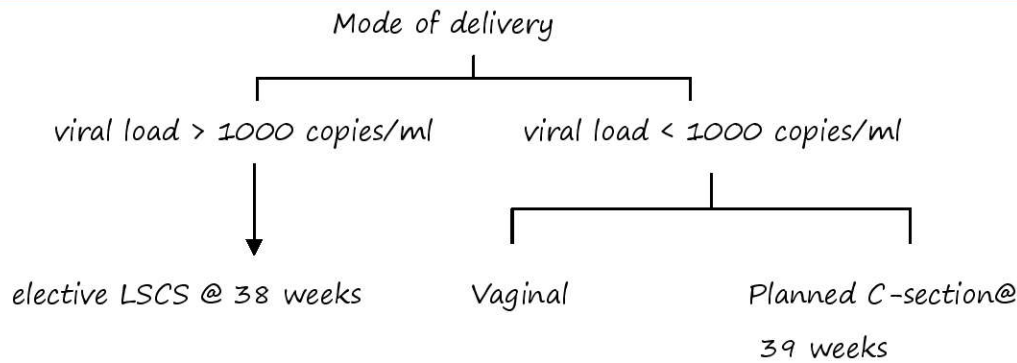
Antepartum care

00:11:05

- Initiate ART as early as possible
- If RNA levels >500 – 1000 copies/ml
- ↓
- Order for drug resistance
- ↓
- Change if needed.
- Repeat viral copies every 2-4 weeks
- ↓
- Viral copies monthly until RNA undetectable
- ↓ Once undetectable
- Check Viral copies 3 monthly
- CD₄ count → every 3-6 monthly
- Lifelong ART – Irrespective of CD₄ count/viral load

Intrapartum care

00:14:45



- If patient presents in labour / planned for section.



IV zidovudine – starting 3hrs prior to scheduled C-section

- If patient is on **ORAL ART** → Take tablet with sips of water even on the day of delivery
- If undergoing C-section for obstetric indication, then only at 39 completed weeks.

Do's and Dont's

00:19:32

- During labour

Better avoid	Dos
Artificial rupture of membranes (ARM)	Augmentation with oxytocin (If viral load < 1000 copies)
Scalp Electrode	Early cord clamping
Episiotomy	Neuraxial analgesia
Instrumental delivery	

- **During Postpartum**

- ART should be continued life long
- Breast feeding
 - Should be continued in developing countries
 - Withheld in developed countries.



Zika virus

00:39:50

- Primary mode of transmission is mosquito bite
- sexual transmission is also possible
- Cause → Gillian Barr syndrome
- fetus can be severely infected even mother is asymptomatic
- fetus infection rate > 6 %
- Birth defect in > 5-15 % among infected fetus

Congenital zika syndrome

Features

- microcephaly – intracranial calcifications
- lissencephaly – ocular abnormalities
- ventriculomegaly – congenital contractures

Treatment:

- No specific treatment
- No vaccine available

Malaria in Pregnancy

00:42:10

- Pregnancy → increase susceptibility to malarial infection
- Severely pf infection more during pregnancy

Effects of malaria on pregnancy

- Increase still birth – IUGR
- Preterm labour – Anemia

- Abortion
- Incidence of congenial malaria → < 5%

Treatment during pregnancy

- DOC for all uncomplicated cases – chloroquine
- Chloroquine resistant cases – mefloquine
/quinine/sulfoxide +
pyrimethamine
- DOC for falciparum cases – artesunate

Tuberculosis in pregnancy

00:44:15

- Increase Risk of Preterm delivery
Low birth weight
Growth restriction
Pre- eclampsia
- Treatment of latent infection: INH – 300 mg OD
X 6-9 months after 1st trimester
- Treatment of active disease: 2HRZE+ 4HR
- If HIV positive lady, patient on ART
start with ATT
↓ 2-4 weeks
Starts HAART
- C/I during pregnancy → aminoglycoside
Streptomycin (ototoxicity)

Breast feeding

- Only women on ATT → continue breast feeding
- Both mother & baby on ATT → No breast feeding (to avoid toxicity)
- Women with active disease and not on ATT → avoid breast feeding Isolate the baby & Treat baby with INH 10-20 mg /kg/day for 3 months

Vaccination

BCG

→ should be given as early as possible.

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TUTORIALS

LOGIN FOR SUCCESS

RUBELLA IN PREGNANCY

Rubella During Pregnancy

00:01:39

- 25-50% of infected pregnant women – Asymptomatic
- If symptomatic, presents with
 - Maculopapular rash: begins in face and spreads to trunk and extremities
 - Fever/sore throat/Arthralgia/Joint pain
 - Post auricular lymph node/Conjunctivitis/ Headache
- Incubation period: 12-23 days
- Viremia usually precedes clinical symptoms by about a week
- Viremic = infectivity
- Infective through 7 days after the rash.
- Subclinical infections → congenital rubella syndromes.

Transmission rate	Fetal infection	Congenital defect	Miscarrying rate
1 st 12 wks.	90%	85%	25%
13 th /14 th wks.	50%	35%	-
After 28 wks	25%	-	-
>36 wks.	100%	-	-

- It leads to
 1. Cardiac septal defects – VSD
 - PDA/PS
 2. Microcephaly – Hepatosplenomegaly
 3. Cataract – SNHL
 4. Micro-ophthalmia – Low IQ
 5. Retinopathy – Neonatal thrombocytopenic purpura
 6. Glaucoma – radiolucent bone disease

Long term effects

1. Panencephalitis
2. Insulin dependent diabetes
3. Thyroid disorders
4. Precocious puberty

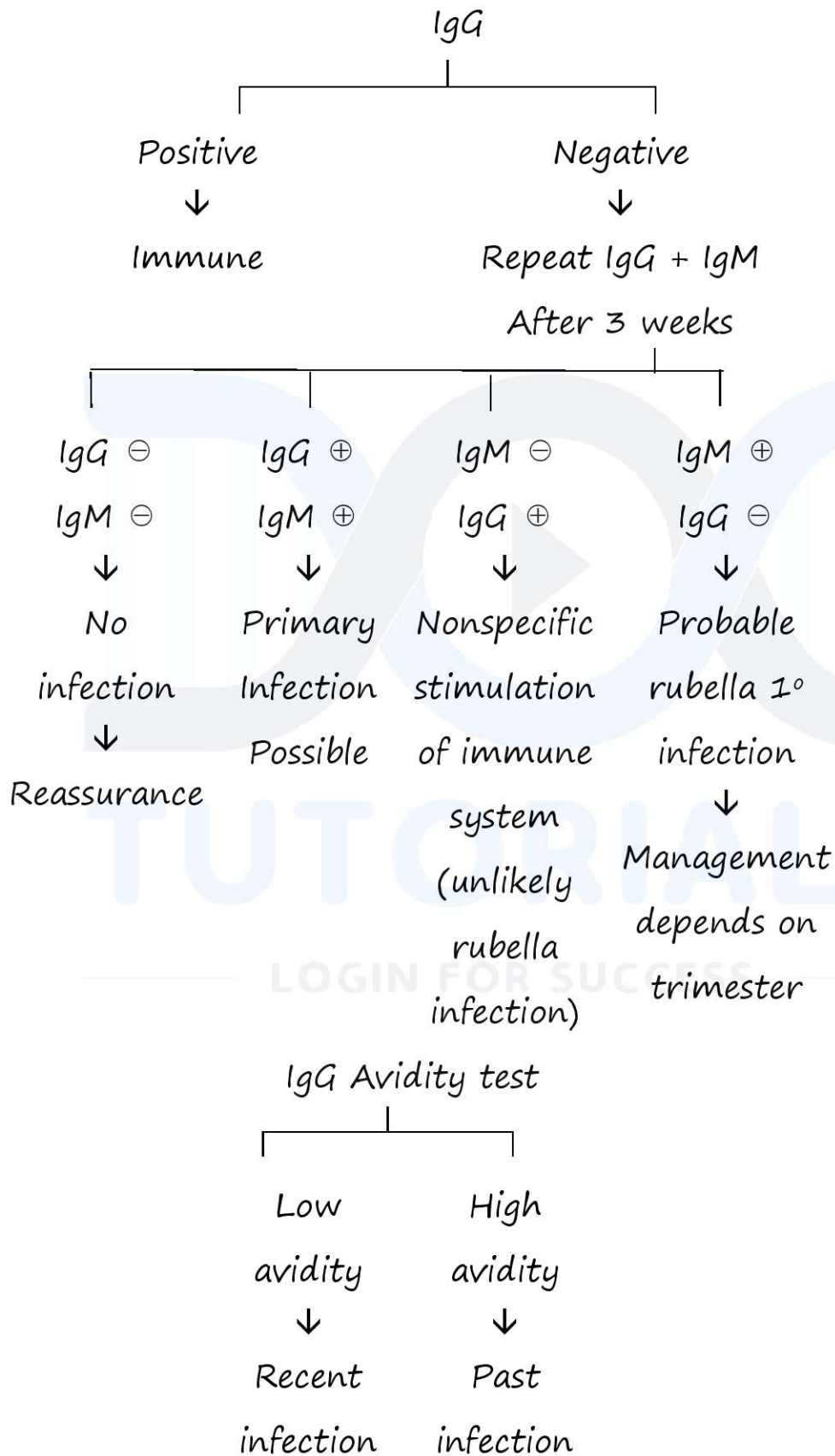
Antibody response

- IgM → appears 4-5 days after the onset of rash
- IgG → appears 1-2 wks. after the onset of rash
- IgG avidity test → high avidity (2 months)
- IgG titer → 4-fold increases in IgG over a period of 2 weeks.

Screening of exposed women

00:19:42

- If asymptomatic but exposed to rubella



Prenatal Diagnosis of Fetal Rubella Infection:

- Fetal blood (Cordocentesis)
- Amniotic fluid (Amniocentesis)
- Placenta (Chronic villus biopsy)

IgM:

- PCR
 - Best option for amniotic fluid

Role of Passive Immunization

00:33:33

- Immunoglobulins
- ACOG → Yes, Ig
- CDC → Ig are not helpful; neither protect mother nor fetus false sense of reassurance
- Who should receive ? → Not willing for termination of pregnancy
 - 20ml i.m. within 72 hours

LOGIN FOR SUCCESS

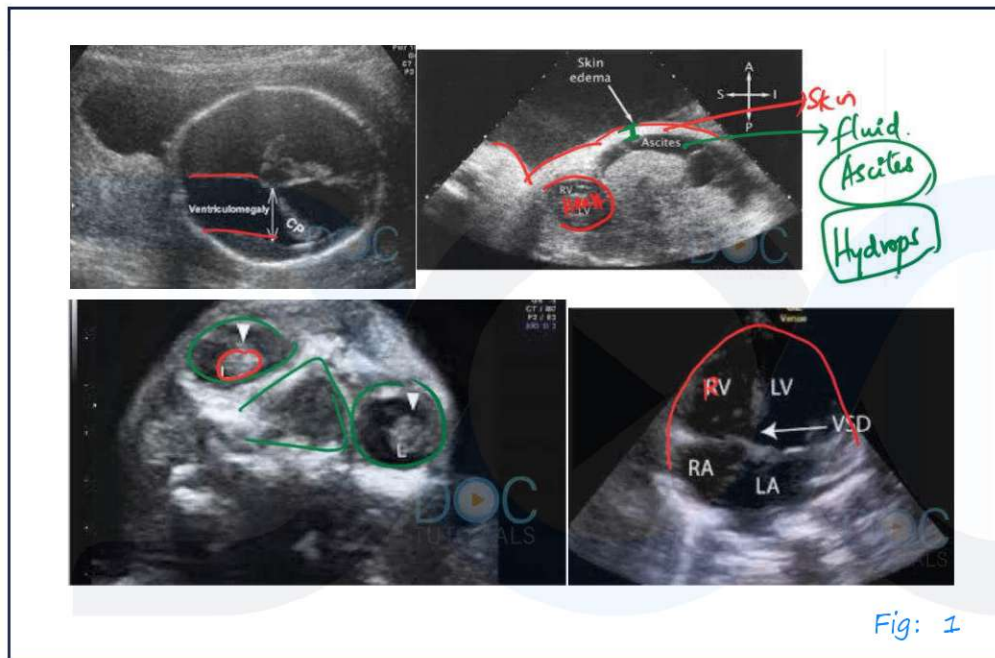
USG Features of Congenital Rubella Syndrome

00:36:40

Long term effects

1. Ventriculomegaly
2. Microcephaly
3. Intracranial calcification/cyst
4. Cataract
5. Micro-ophthalmia

- 6.ASD/VSD/PDA
- 7.HSM
- 8.Hydrops
- 9.Hyperechogenic bowel polyhydramnios
10. Placentomegaly
 - IUGR/IUD/Still birth



Management Guidelines

00:41:36

- < 12 weeks → Termination of pregnancy
- 12-18 weeks → Prenatal diagnosis of fetal infection
- AF PCR
 - If positive → Terminate
 - If negative → Surveillances USG monitoring
- Beyond 18 weeks
 - Continue pregnancy USG monitoring for features of CRS neonate screening - IgM

Routine Antenatal Screening

00:47:00

- Yes, IgG rubella
- IgM positive → Top

Adequate Proof of Rubella Immunity:

- i. 1 dose of rubella vaccine
- ii. Lab c/o IgG

Vaccination in Pregnancy

00:49:02

- Live attenuated vaccine
- Strictly c/I during pregnancy
- Ideally given in pre-conceptional → ideal to postpone pregnancy – 1 month after getting a shot vaccine
- 2.6% risk → Congenital rubella infection if vaccine given in 1st trimester
- No termination of pregnancy, if vaccine is accidentally administered during pregnancy

LOGIN FOR SUCCESS

FIBROID IN PREGNANCY

Fibroid in Pregnancy

00:00:36

- Incidence – 2%
- Highest in – black women
- Growth factor → progesterone > Estrogen
- Pregnancy → increases progesterone



Fibroids – Grow/increases size

- Regress
 - Remain unchanged
- IOC → USG
 - Gold standard → MRI

Symptoms

00:03:31

- Asymptomatic (most)
 - Acute/Chronic pain → pressure
- ↓
- Degeneration
- Red degeneration/corneous degeneration
 - Due to hemorrhagic infarct
 - Not an infective condition
 - Acute focal abdominal pain/tenderness
 - Low grade fever
 - Increases ESR
 - Leukocytosis
 - Vomiting

- D/D for red degeneration
 - Appendicitis
 - Placental abruption
 - Ureteric calculus
 - pyelonephritis

Treatment

00:07:54

- Conservative
- Hydrate
- Analgesics
- Bed rest
- Self-limiting condition resolves

Pregnancy complications with fibroids 00:09:27

1. Preterm labour
2. Placental abruption
3. Malpresentation
4. Obstructed labour
5. Dysfunctional labour
6. C - section
7. PPH
8. Abortion
9. IUGR
10. Pl. Previa

Treatment of Fibroid in Pregnancy

00:11:16

- Conservative
- Hydration
- Analgesics
- Antibiotics
- Bed rest
- Only fibroid that can be removed during pregnancy – pedunculated sub serous fibroid
- Infection → more common in post-partum period.



OVARIAN TUMORS IN PREGNANCY

- Incidence 1 in 100 – 1 in 200

Types

00:00:50

- Corpus luteal cyst
 - Endometrioma
 - Benign cystadenoma
 - Mature cystic teratoma
- 1% are frankly malignant

Symptoms

00:02:09

- Mostly asymptomatic
- Acute pain – torsion/ rupture/ hemorrhage
- Chronic pain

Diagnosis

00:02:57

- IOC – USG
 - Gold standard – MRI
- Ca125 – elevated in 1st trimester & post-partum,
2nd & 3rd trimester – normal

Management

00:04:49

- Symptomatic

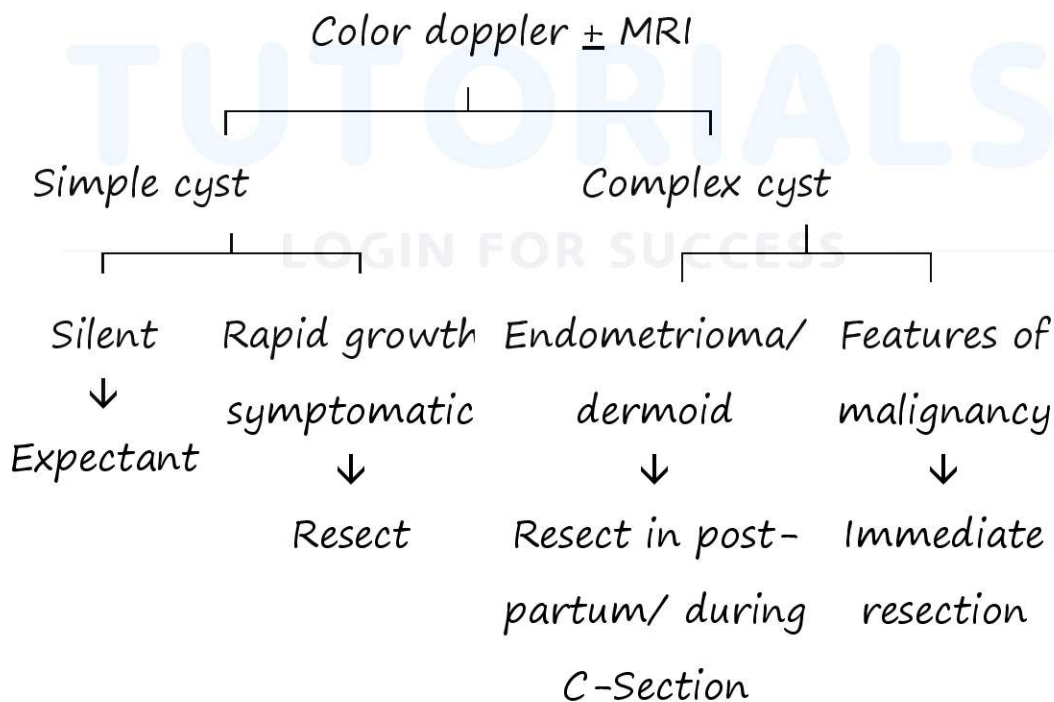
- Most common cause of ovarian hemorrhage
- Rupture of corpus luteal cyst.

If Salvageable ovary ↓ Untwist ↓ Remove only cyst	If Persistent cyanosis ↓ Remove adnexa Cyst/remove FT & ovary
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- Asymptomatic

- Cyst < 5cm → likely to be corpus luteal cyst resolves by early 2nd trimester expectant management.

-Cyst/mass (5-10 cm) -



- Cyst ≥ 10cm - Immediate resection

DRUGS IN OBSTETRICS

Risk Classification/ Safety Rating System

- Drugs are classified into five categories

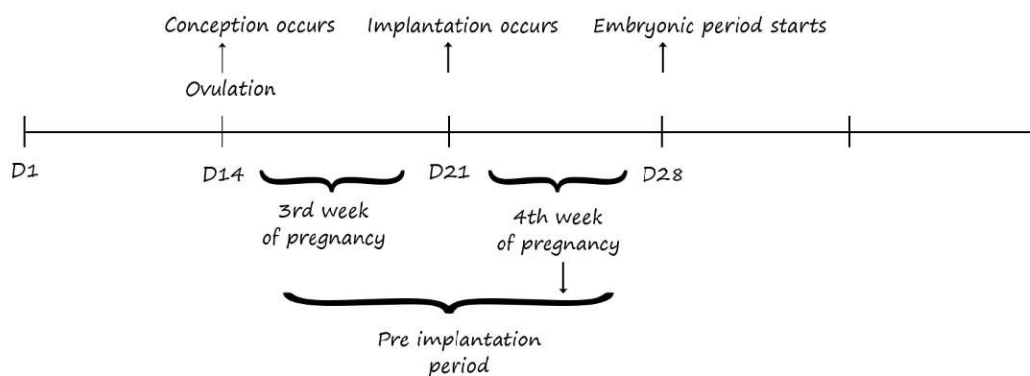
Categories		Animal Studies	Human Studies
A	Safely established	⊕ No risk	⊕ No risk
B	Safety likely	⊕ No risk Risk +	Not available ⊕ No risk
C	Teratogenicity possible	⊕ Risk + Not available	Not available Not available
D	Teratogenicity Probable	Risk ⊕	Risk ⊕
X	Teratogenicity likely	Risk ⊕	Risk ⊕

- D - Benefit > Risk
- X - Risk > Benefit

Category X – Drugs

1. Androgen & testosterone derivatives
2. ACE inhibitors/ARBs
3. Coumarin derivatives (eg: warfarin)
4. Carbamazepine
5. Diethylstilbestrol
6. Folate antagonist (methotrexate/aminopterin)
7. Lithium
8. Phenytoin
9. Primidone
10. Statins
11. Streptomycin & kanamycin
12. Tetracycline
13. Thalidomide/leflunomide
14. Trimethadione/paramethadine
15. Valproate
16. Vitamin A and its derivatives (eg: isotretinoin)

Trimester based risk



i. Preimplantation period

- 1st 2 weeks after conception
- All or none period (either miscarriage or no damage at all happens)

ii. Embryonic period

- From 3rd to 8th week post conception
- Period of organogenesis (maximum a teratogenicity)

iii. Fetal period

- Beyond 8wks of post conception
- Certain organs remain vulnerable (external genitalia)

Effects of Radiation

1. IUGR

2. Anomalies

3. Impaired brain function

4. Malignancies

- Effect depends on the GA of dose of ionizing radiation.

i. First 14 days (2wks after conception) –

- All are non period
- $> 0.1\text{Gy}$ or $>10\text{ rad}$ → death of embryo/miscarriage
- $<0.1\text{Gy}$ or $<10\text{ rad}$ → No effect

ii. At all GA

< 0.05 or $<5\text{ rad}$ – no adverse effect at all

iii. After 16wks

<0.5 Gy or <50 rad – No effect

- The following imaging studies have no effect on fetus even if exposed in embryonic period
 - i. Diagnostic X ray of head, neck, chest, limbs
 - ii. USG/MAI
 - iii. Dental X ray
 - iv. CT scan not involving the abdomen of pelvis
- Lead apron must be used to minimize fetal exposure to avoid abdominal exposure.
- Women imaging in imaging studies & must use lead apron and dosimeter.

Teratogenic drugs

i. Alcohol

- ≥ 6 drinks/wk for ≥ 2 wks
- ≥ 3 drinks per occasion for ≥ 2 to occasions
- Binge drinking is more dangerous i.e., continuous exposure on daily basis.

Fetal Alcohol Syndrome (Diagnostic criteria)

1. Dysmorphic facial features –
 - a. Short palpebral fissure
 - b. Thin vermilion border of upper lip
 - c. Smooth philtrum
2. Prenatal/Post natal growth impairment ($<10^{\text{th}}$ centile)

3. Abnormal brain growth

- a. Head circumference $\leq 10^{\text{th}}$ centile
- b. Structural brain abnormalities
- c. Recurrent non febrile seizures.

4. Neurobehavioural impairment –

Developmental delay/global cognitive impairment

Other alcohol related birth effects

- Cardiac – ASD/VSD/Aberrent great vessels
- Skeletal – Radioulnar synostosis, joint contracture, scoliosis.
- Renal – Renal agenesis/hypoplasia/dysplastic kidney/horse shoe kidney.
- Eyes – Strabismus, ptosis, optic N. hypoplasia
- Ears – Conductive/sensory neural hearing loss
- Omphalocele/gastroschisis.

Antiepileptic drugs

00:37:14

- Highest risk – valproate
- Other dangerous drugs – Carbamazepine
 - Phenytoin
 - Phenobarbital
 - Topiramate
- Usual anomalies – Orofacial clefts
 - Cardiac malformations

- Most important anomalies neural tube defects – mainly valproate

Low IQ

Poor cognitive development

Fetal Hydantoin Syndrome

00:38:53

- Seen in fetal exposure to phenytoin
- Features – Upturned nose
 - Mild midfacial hypoplasia
 - Long upper lip with thin vermillion
 - Distal digital hypoplasia
- Safer antiepileptic's – i. Leviteracitam
 - ii. Lamotrigene

Warfarin

00:41:08

- Anticoagulant
- Vitamin K antagonist, long t $\frac{1}{2}$
- Low molecular weight readily crosses placenta

Warfarin embryopathy

- Nasal hypoplasia, depressed nasal bridge, choanal atresia
- Stippled epiphysis of femur, humerus, calcaneum, distal phalanges
- Hemorrhage in fetus

CNS

- Agenesis of corpus callosum
- Cerebellar vermain agenesis
- Micro opthalmia
- Optic atrophy, blindness
- Deafness
- Developmental delay
- Critical GA for warfarin embryopathy 6-9 wks
- Incidence at warfarin embryopathy among exposed fetus is 6%

Lithium

00:44:45

- Ebsteins anomaly – Displacement of tricuspid valve
Tricuspid regurgitation
Marked RA enlargement
- Incidence among exposed - $\frac{1-4}{1000}$
- Neonatal lithium toxicity
 - Due to exposure near delivery
 - Drugs should be decreased or stopped 2-3 days prior to delivery
- Features of neonatal lithium toxicity

- Hypothyroidism - Diabetes insipidus - Cardiomegaly - Bradycardia	}	Persits for 1-2 wks after delivery and subsides later.
---	---	---

- ECG abnormalities
- Cyanosis/hypotonia

SSRI

00:48:51

- Not all SSRIs are teratogenic

CNS

- Most teratogenic SSRI
- Cardiac anomalies (ASD NSD) – most common system involved
- Neonatal behavioural syndrome (jitteriness, irritability, hypo/hypertonia, feeding abnormality, vomiting, hypoglycemia, thermoregulatory instability, respiratory problem)
- ASD is the most common anomaly.

Anticancer Drugs

00:51:10

- i. Cyclophosphamide – Leads to
 - Abortion
 - Limb defect
 - Cleft palate
 - Eye abnormality
 - Developmental delay
- ii. Methotrexate
 - Potent teratogen, abortifacient
 - Used in ectopic pregnancy and for MTP

Fetal Methotrexate aminopterin syndrome:

- Occurs when complete termination of pregnancy doesn't happen
- Fetus is most vulnerable between 8-10 weeks
- Critical dose is 10 mg/wk
- But for termination of pregnancy the dose given is 50 mg/m²
- So when, ectopic pregnancy treated with methotrexate later turns out to be intra uterine, then terminate the pregnancy because the dose given in treatment of ectopic pregnancy is much higher than the critical dose of methotrexate leading to anomalies in fetus.

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Features of fetal methotrexate aminopoterin syndrome

- Craniosynostosis**
 - Clover leaf skull
 - Wide nasal bridge
 - Low set ears
 - Micrognathia
 - Limb abnormalities
- Cardiac anomalies – conotruncal defects**

Tamoxifen

00:59:15

- SERM
- Cause, DES like syndrome, vaginal adenosis

Trastuzumab

01:00:07

- Oligohydromnios
- Pulmonary hypoplasia
- Renal failure
- Skeletal abnormalities
- Neonatal death

DES (Diethyl Stilbestrol)

01:01:41

- Clear cell adeno carcinoma of vagina of cervix.
- Incidence 1/1000 exposed fetuses)
- Features in female
 - Genital tract abnormalities
 - Hypoplastic/T shaped uterine cavity
 - Cervix collare, hood, septa, coxscomb cervix.
 - Withered fallopian tube
 - Premature menopause
 - Breast cancer
- Features in male
 - Epididymal cyst
 - Microphallus
 - Hypospadias
 - Cryptorchidism

Corticosteroids

- Increases risk of cleft if given before 24 wks of GA
incidence 3/1000 exposure

Prednisone

- Prednisolone is active metabolic of prednisone
- Prednisolone is inactivated by 11β HSD in placenta



Less prednisolone reaches fetus

Mycophenolate

- Potent teratogen/abortifacient
- Anomaly rate – 30%
- Abortion rate – 30%
- Microtia, auditory canal atresia, cleft coloboma
- Short finger, hypoplastic nails
- Cardiac defects

LOGIN FOR SUCCESS

GESTATIONAL TROPHOBLASTIC DISEASE (GTD)

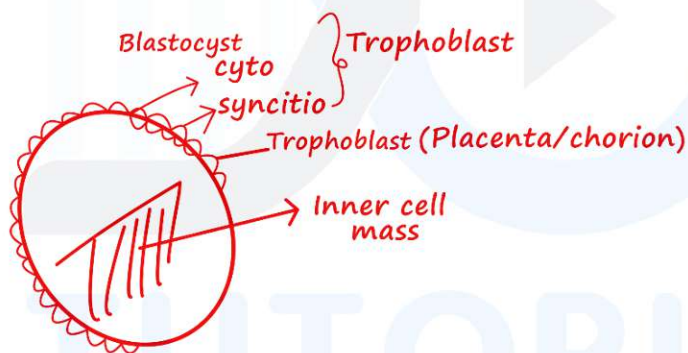
Definition:

00:01:07

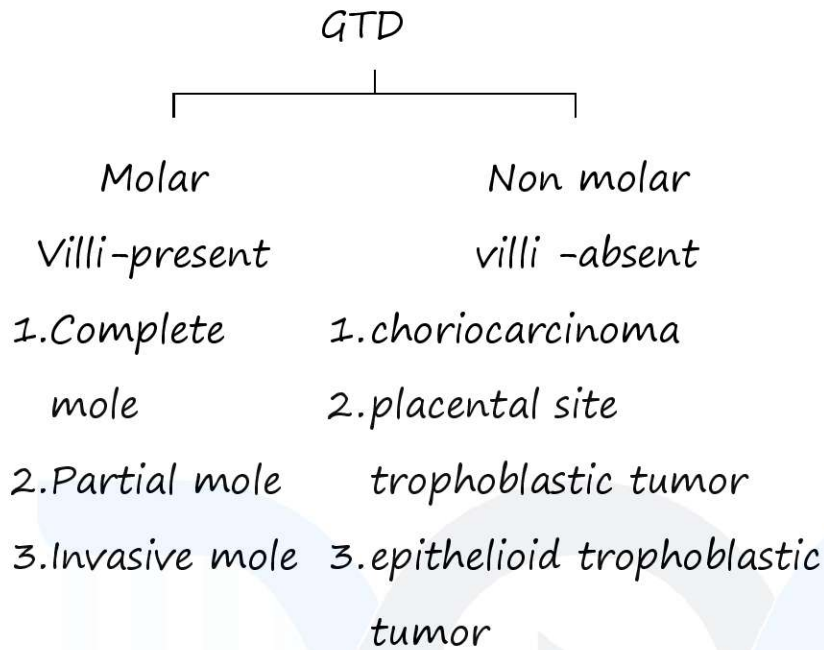
- Gestational trophoblastic disease includes a group of disorders characterized by abnormal trophoblastic proliferation
 - I. Uncontrolled
 - II. Degenerative tissue

Pathophysiology:

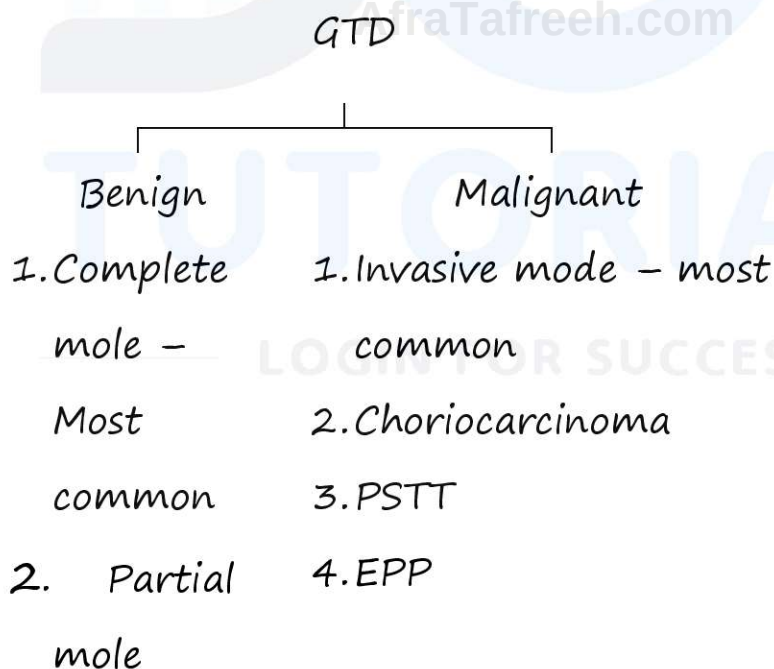
00:02:44



- Abnormal -uncontrolled proliferation of tissue
Trophoblast fills the uterus
- Inner cell mass → degenerative
- Complete degeneration inner cell man → complete mole
- Partial degeneration of Inner cell mass → partial mole



Other Classification



Incidence & risk factor:

00:10:58

- Incidence → 1-2/1000 deliveries
- Country with highest incidence → Philippines → 1 in 80
- India → in 400

Risk factors

- Asian population
- Extremes of age → teenage, elderly
- Past H/p molar pregnancy
- Vitamin A deficiency
- AB Blood group

Women with age 36-40 → 3-fold increase risk of GTD

> 40 years → 10-fold increase risk of GTD

Pathogenesis

Complete mole → completely trophoblastic

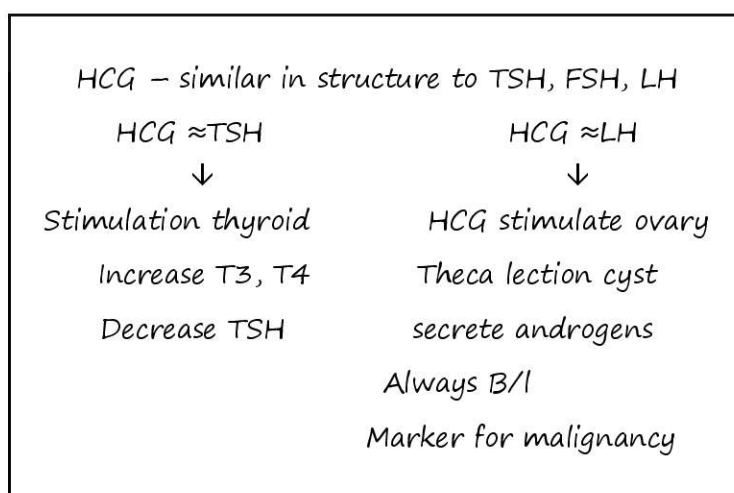
Completely paternal

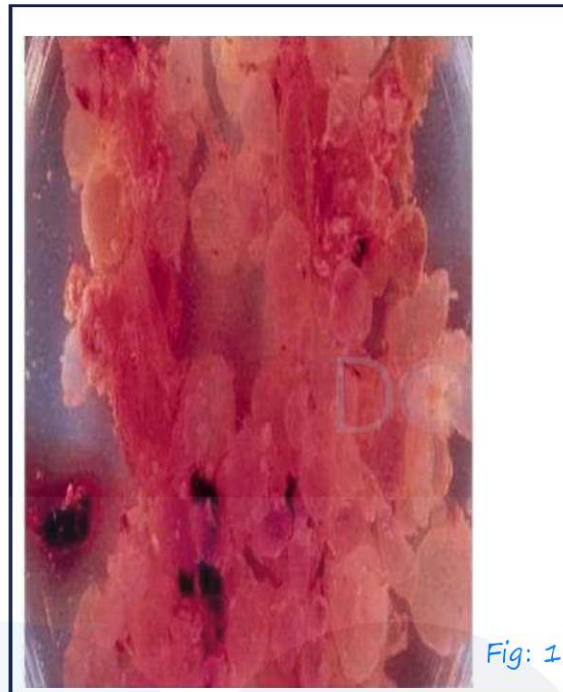
<u>Complete mole</u>	<u>Partial Mole</u>
• Completely trophoblastic • Completely paternal • Empty egg + 1 sperm 23x/y	• partially trophoblastic • partially paternal/maternal • egg 23x + 2 sperms
↓ Multiply	↓
Androgenesis (most common)	Dispermy/Diandry
Diploid 46xx/46xy (or) 46yy	23x + 23X 23 X 23y
Empty egg + 2 sperms	23x 23y 23y
↓ Multiple	┌──────────┐
Dispermy/diandry (20%)	69xxx 69xxy 69xyy
23 x 23 x 23 y	↓
23 x 23 x 23 y	most common
Diploid 46 xx 46xy 46 YY	
↓	
most common	

Clinical features

00:26:58

- Most common presenting symptom → vaginal bleeding
- Other symptoms → amenorrhea. pain
- Grape like vesicles p/v (hydropic change)
- H → hemorrhage
- H- High levels of HCG
- H- Hypertension (Therefore, HCG is similar to TSH, ESH, LH)
- H- height of uterus (bigger for given gestational age – complete mole)
- Hyperemesis
- Hyperthyroidism
- Small for given gestational age – partial mole
- Uterus → soft / doughy
- Anemia
- Respiratory distress
- Sepsis
- Perforation
- DIC





Important Table –Pearl

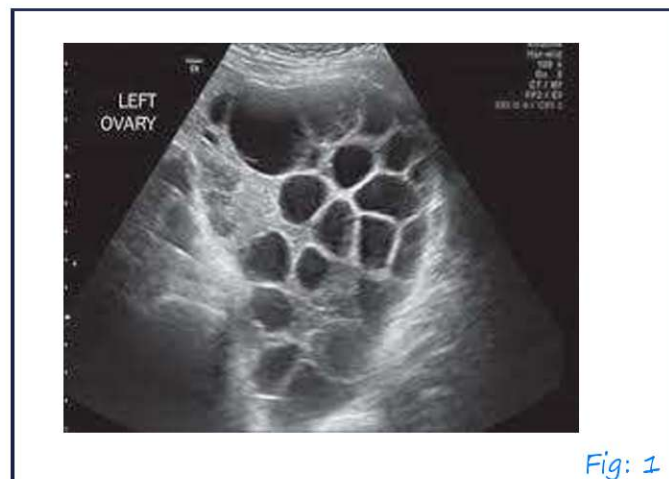
complete mole	Partial mole
HCG → > 1 lakh	< 1 lakh
UDG → Mixed echogenic multiple cystic spaces	Multiple cyst space + Placenta + Fetal parts (Congenital anomalies IUGR)
karyotyping → 46XX/46xy	69 XXY
Immunostaining P57kip-2 → Negative	Positive
HPE → fetal parts – Absent	Fetal parts – Present
Snow storm appearance	

 Complete mole	 Partial mole
Medical complication: +/-	-
Malignant potential	
Persistent GTD 15-20percent	1-5 percent

Theca lutein cyst

00:42:57

- B/l ovarian cysts
- Hyperstimulation of ovaries by HCG
- Seen in normal pregnancy, multiple pregnancy, DM
 - If >6cms is a risk for malignancy
 - It secretes Androgens leads to Virilization.
 - Observation-Torsion/hemorrhage/rupture- USG guided aspiration.



Diagnosis:

Clinical	HCG	USG	Karyotyping (P57Kip2)	Immunostaining +HPE
Complete	>1lac IU	Mixed echogenic Multiple cystic spaces	46xx	Negative + fetal parts absent
Partial	<1lac IU	Mixed echogenic Multiple cystic spaces+placenta + fetal parts+congenital anomalies+IUGR	69xxy	Positive + fetal parts present



Fig: 2

Management:

00:54:56

Surgical – suction evaluation

Contraversies

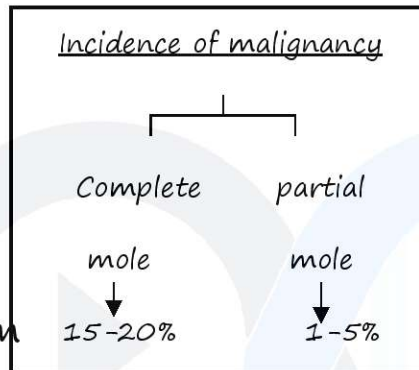
			ACOG	GTG
i.	Intra operative oxytocin	-	✓	X
ii.	Other uterotonics	-	✓	X
iii.	Anti- D	-	✓	✓
iv.	Prophylactic chemotherapy	-	No role in presenting malignancy	

Indication for Hysterectomy

1. Age > 40 years
2. Perforation
3. Hemorrhage
4. Completed family

Risk factors or malignancy:

1. Complete mole
2. Old age
3. HVG $\rightarrow$ >1 lakh
4. Increased uterine size
5. Theca lutein cyst > 6 cm
6. Slow fall in HCG levels



Follow up:

Before evacuation - Chest x-ray, β -HCG

Blood grouping

CBC, LFT, RFT

Viral markers

Thyroid profile

After evacuation → β -HCG within 48 hrs. of evacuation

↓
Weekly until 3 Negative HCG [partial - 7 wks.
complete - 9wks]

↓
Monthly for 6 months

↓
Stop

Contraceptive of choice: Hormonal → Combined oral
contraceptive best

- When to start - After HCG - becomes Normal

Gestational Trophoblastic Neoplasia (GTN)-

01:11:21

Types

- i. Invasive mole - M/C
- ii. Chorio carcinoma
- iii. PSTT
- iv. ETT

GTN

- 50% of GTN - follow Molar pregnancy
- 25% of GTN - occur following abortion
- 25% GTN - occur following N pregnancy

Criteria for diagnosis

- i. Plateau of HCG ($\pm 10\%$ of previous value) 4 values in 3 wks.
- ii. Increasing HCG ($> 10\%$ of previous value) 3 values in 2 wks.
- iii. HCG remaining undetectable range ≥ 6 months
- iv. Histologic criteria for GT

Invasive Mole (or) chorioadenoma destruens	Choriocarcinoma
→ always arises after molar pregnancy → Never after N pregnancy → Villi → present → locally invasive → Does not spread to distant organs	→ Mostly after N pregnancy → 1/3 rcl of them after Molar pregnancy → Villi → absent → spread to distant organs → Routine of spread is Hematogenous → m/c site of Metasis → lungs

PSTT and Epithelioid tumors:

01:19:30

- Malignant varieties
- Arise from cytotrophoblast (Intermediate trophoblast)
- Moderate increase in HCG
- Tumor marker – HPL (Human Placental Lactogen)
- Chemo resistant
- Treatment of choice – hysterectomy
- Locally invasive

Staging:

- I. Limited to uterus
- II. Involves extrauterine pelvic structure (ovaries /ligaments)
- III. Lung involvement
- IV. Distant organs (Brains/liver)

Modified WHO scoring

Parameter	0	1	2	4
Age (Y)	<40	≥ 40	---	---
Antecedent pregnancy	Mole	Abortion	Term	---
Interval in months from index pregnancy & chemotherapy started within	<4 months	4-6 months	7-12 months	>12 months

Pretreatment b-hCG level	$<10^3$	$10^3 - 10^4$	$10^4 - 10^5$	$>10^5$
Largest tumor (CM)	<3	3-4cms	≥ 5	
Site of metastases	---	Spleen kidney	Gastrointestinal	Brain, Liver
Number of metastases	---	1-4	5-8	>8
Prior chemotherapy	---	---	Single	>2

- Score $\rightarrow \geq 7 \rightarrow$ multiagent chemotherapy
- Score $\rightarrow \leq 6 \rightarrow$ Single agent chemotherapy

Treatment and follow up:

- Single agent chemotherapy drug $\rightarrow$ Methotrexate
- Multiagent chemotherapy $\rightarrow$ EMACO

E – etoposide

M – methotrexate

A – actinomycin

C – cyclophosphamide

O – Oncovin

Cure rate: 100% for who's treated with single agent

94 % $\rightarrow$ for who's treated with multiple agent

Subsequent pregnancy: Placenta $\rightarrow$ sent for HPE

HCG $\rightarrow$ sent @ 6 weeks post-partum

GTD- RCOG updates 2020

01:31:16

- Most common symptoms → abnormal vaginal bleeding
- Hemoptysis /seizures → GTN can be a presetting symptom
- Suction Evacuation → better to do under ultrasound guidance
- Partial mole Treatment → medical methods can be done (Fetal parts present)
- Anti -D prophylaxis → given for partial & complete mole
- Pre evacuation
(Cx) preparation → Yes, can be done
Just prior to evacuation
- Oxytocin infusion → can be better avoided
- Repeat surgical removal: In case of retained products
- Routine HPE:
 - Complete mole → presents with classical symptoms
→ molar pregnancy → sent for HPE
 - Partial Mole → Abortion (missed)
→ if fetal parts seen → not need to sent for HPE
→ if fetal parts not seen → sent for HPE
- Follow up after miscarriage – MTP/ spontaneous abortion

(or)

Complete abortion

- UPT after 3 wks. of miscarriage

- To make sure there is no residual HCG activity
- Elevated HCG after a possible (PUL) pregnancy of unknown location

2 doses of methotrexate



HCG SLL high



Possible GTD

Evaluated accordingly

- Persistent vaginal bleeding after a pregnancy
→ wait for 8 wks.



HCG – persistent



Possible GTD

- Biopsy of secondary deposits in vagina – No

Should not be done

Risk of hemorrhage

- Ectopic molar pregnancy

Treat ectopic according to ectopic guidelines

Salpingectomy /salpingostomy

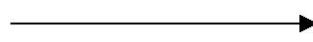


Follow up – HCG values

- Combined molar pregnancy & viable Twin

Risk of perinatal morbidity/mortality → high

Risk of GTN



high

- Typical & atypical placental site nodule

Typical

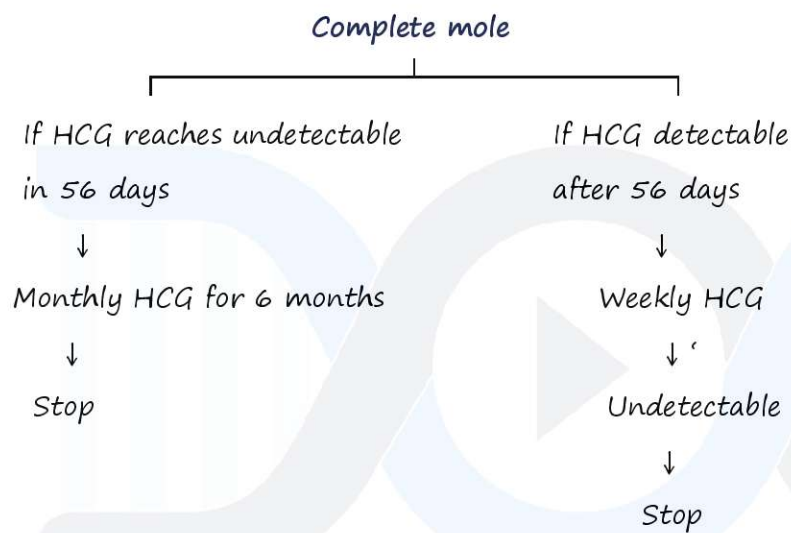
Atypical

Only follow up

Risk of GNT f/b with HVG

Follow up

01:56:01



Portal mole

- Need not be follow up for 6 months
- 2 negative hcg value 4 weeks apart
- Subsequent pregnancy – Interval

Only suction evacuation → 6 months from Negative HCG

No chemotherapy

If chemotherapy → 1 year from completion of chemo

- Subsequent pregnancy – HPE of placenta

Need not be done in every case

No chemo – No HPE

- Subsequent pregnant -HCG

No chemo – No routine HCG

If chemotherapy ✓ → HCG follow up needed in subsequent Pregnancy

Effect of chemotherapy

- Chemotherapy leads to premature menopause
- Chemotherapy adversely effects fertility

HRT

- Can HRT be given? → Yes
- When to start HRT → once HCG levels are N

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TUTORIALS

LOGIN FOR SUCCESS

HYPEREMESIS GRAVIDARUM

Nausea and vomiting in pregnancy: 00:00:25

- **Incidence: 80%**
- Time of onset → begins in 1st Trimester → 6-8 weeks
Peaks → 10 weeks
Disappears → 18-20 weeks

Hyperemesis: 00:02:14

- Incidence: 0.3-3.6 %
- Definition:
 1. Loss of weight -at least 5% pre pregnancy due to vomiting
 2. Electrolyte imbalance/ketosis
 3. Dehydration

Hormones responsible are:

1. HCG
2. Steroids → estrogen & progesterone
3. Cortisol
4. Thyroxine

Condition associated with Hyperemesis:

1. Multiple pregnancy
2. Molar pregnancy
3. Chronic carcinoma

4. Rh incompatibility

5. Diabetes

PUQE scoring / Index

00:09:11

1. In the last 24 hrs.' how long you felt nauseated →

Not at all	≥ 1 hr	2-3hr	4-6hr	> 6hr
☐ 1	☐ 2	☐ 3	☐ 4	☐ 5

2. In the last 24 hrs.' how many times have you vomited →

No	1-2	3-4	5-6	≥ 7
☐ 1	☐ 2	☐ 3	☐ 4	☐ 5

3. How many times have you had writhing/dry heave? →

No	1-2	3-4	5-6	≥ 7
☐ 1	☐ 2	☐ 3	☐ 4	☐ 5

Minimum score - 3

Maximum score - 15

Management of Hyperemesis gravidarum:

00:17:00

- Choice of Antiemetic drugs:
- 1st line drugs: cyclizine, Prochlorperazine, Promethazine, chlorpromazine
- 2nd line drugs: Metoclopramide, Domperidone, Ondansetron
- 3rd line drugs: corticosteroid

↓

Hydrocortisone - 100 mg twice daily IV

↓↓ f/d

Oral prednisone 40-50 mg daily



Taper & stop

Fluid replacement

00:21:54

- Normal saline with potassium supplement Q
- Clinical → dextrose containing solutions will precipitates

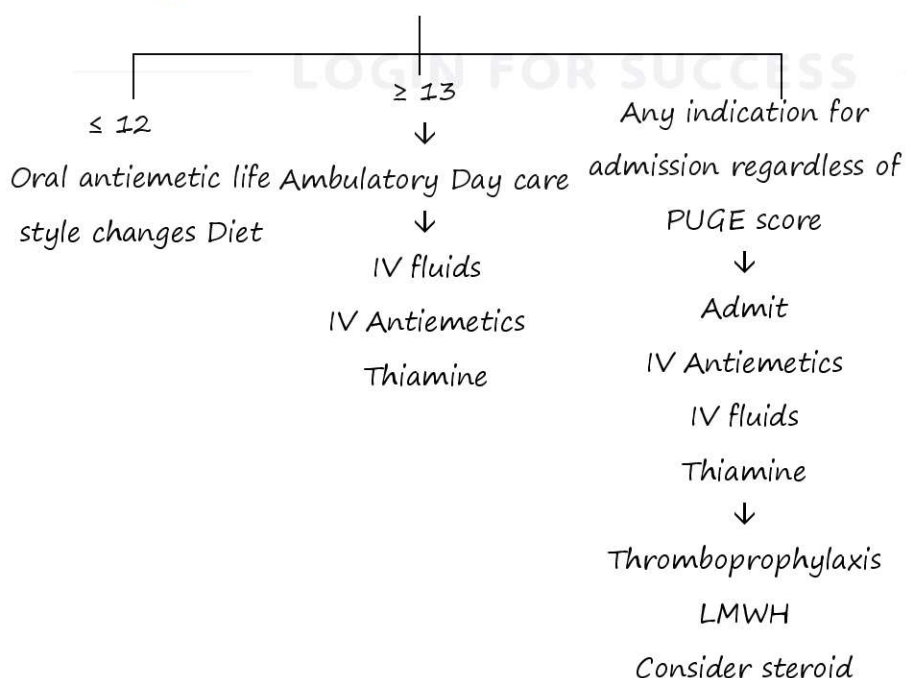
Wernicke's encephalopathy in Hyperemesis Gravidum.

Criteria for Inpatient Management:

00:24:00

1. Continuous Nausea/vomiting not responding to oral antiemetics
2. Weight loss/ketonuria
3. Urinary tract infection – not able to tolerate oral antibiotics

Management based on PUGE SCORE



Supportive drugs:

- Pyridoxine → X
 - Diazepam → X
 - H₂ antagonist ✓
 - PPI ✓
 - Heparin ✓
 - Thiamine ✓
 - Oral Iron preparation X → increase gastric irritation
- Ginger

Hypnosis

Acupuncture

}

✓

Wernicke's encephalopathy:

00:37:40

fatal condition Due to Thiamine deficiency

- Symptoms
 - Confusion
 - Memory loss
 - Ataxia
 - Ophthalmoplegia
- Treatment → High dose Thiamine → 100 mg Iv
- Recurrence rate → 15-80%

THROMBOEMBOLISM DURING PREGNANCY

Venous Thromboembolism (VTE) in Pregnancy

00:00:30

- Relative risk of Venous Thromboembolism (VTE) in pregnancy → Increases by 4-6-fold
- Relative risk of VTE in postpartum period □ Increases by 5-fold
- Incidence of VTE during pregnancy □ 1-2/1000
- Incidence of Antenatal Pulmonary Embolism □ 1.3/10,000

Antenatal Assessment of Risk:

- i. Any previous VTE → High risk - start LMWH prophylaxis (ASAP) (Except a single event related to major surgery)
 - Intermediate risk □ start LMWH prophylaxis (ASAP) Continue throughout pregnancy
- ii. Hospital admission
 - Single VTE related to surgery
 - High risk thrombophilia + NO VTE
 - Medical comorbidities
 - Like cancer, heart failure, active SLE
 - Inflammatory bowel disease, Inflammatory polyarthrititis
 - Nephrotic syndrome

- Type I DM with nephropathy
- Sickle cell disease
- Current IV drug abuse
- Any surgery (Q. appendicectomy)
- OHSS (first trimester)

Other Risk factors

1. Obesity BMI $\geq 30 \text{ kg/m}^2$

2. Age > 35

3. Parity ≥ 3

4. Smoking

5. Gross varicose veins

6. Current pre-eclampsia

7. Immobility (Eg. Paraplegia)

8. Family h/o unprovoked/estrogen provoked VTE in 1st degree relatives

9. Low risk Thrombophilia

10. Multiple pregnancies

11. IVF/ART (Artificial Reproduction Techniques)

- Dehydration (hyperemesis)
- Current systemic Infection
- Long distance travel

≥ 4 Risk factors present
Start prophylaxis LMWH throughout

3 risk factors present
Start prophylaxis LMWH from 28 wks.

< 3 risk factors present
Mobilize patient & avoid dehydration

Postnatal Assessment & Management 00:24:55

High risk factors

- i. Any previous VTE
- ii. Anyone requiring antenatal heparin
- iii. High risk Thrombophilia
- iv. Low risk Thrombophilia + family h/o VTE

Prophylactic dose of LMWH for
At least 6wks postpartum

Intermediate risk:

- Cesarean section in labour
 - BMI > 40 kg/m²
 - Readmission/prolonged admission > 3days
 - Any Surgery (except immediate perineal repair)
- (If >3 risk factors Present consider extending prophylaxis)
- Medical comorbidities (as in Antenatal)

Prophylactic Use of LMWH for at
least 10 days

Low risk

≥ 2 risk factors present
LMWH for 10 days

1. Age >35
2. Obesity – BMI >30kg/m²
3. Parity ≥ 3

4. Smoker

5. Gross varicose vein

< 2 risk factors

Mobilise and hydrate

6. Immobility

7. Current pre-eclampsia

8. Family h/o VTE

9. Low risk thrombophilia

10. Multiple Pregnancy

11. Elective C-section

12. Current Systemic Infection

13. Preterm Delivery (<37wks)

14. Still Birth

15. Mid cavity rotational forceps

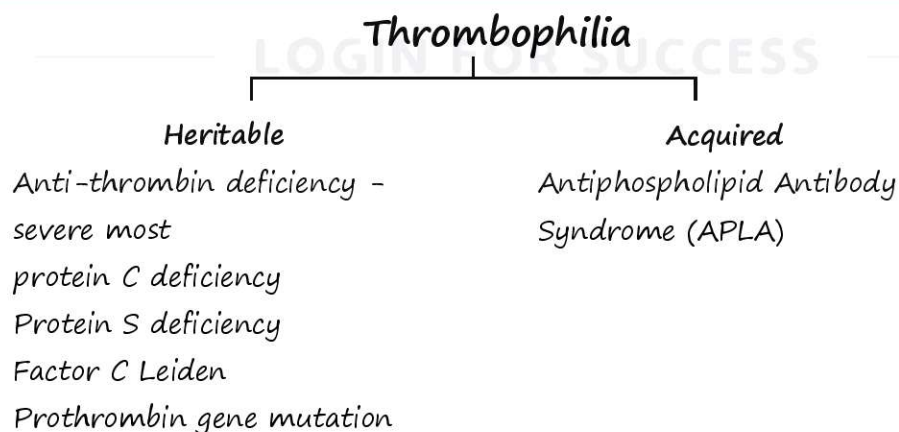
16. Prolonged labour (>24 hrs)

17. PPH > 1 liter of blood

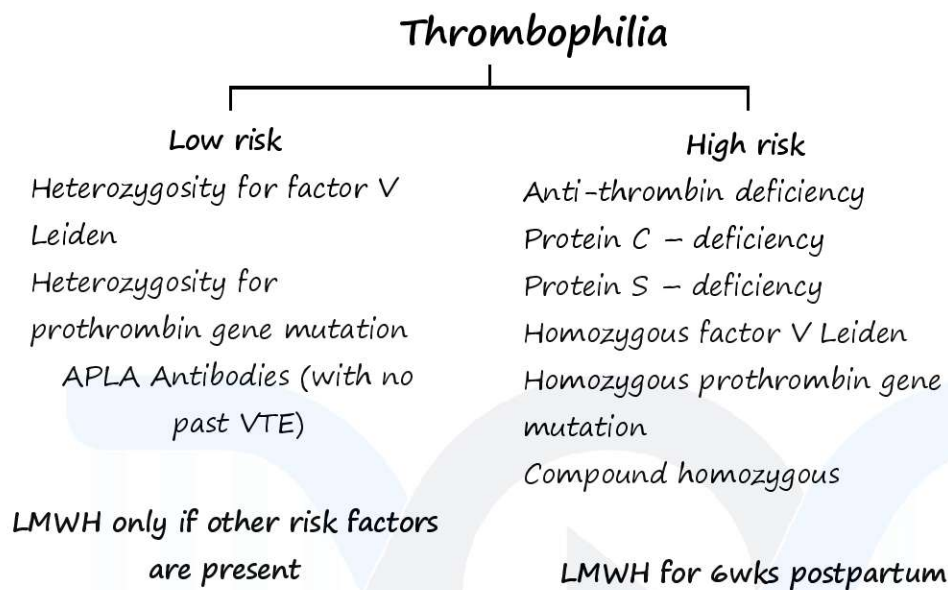
18. Blood transfusion

Thrombophilia

00:39:15



- Antithrombin deficiency and APLA □ Require high dose of LMWH (50% or 75% or full treatment dose to be given)



When to test for Thrombophilia

00:48:55

- Inherited Thrombophilia testing is only for women with family h/o of Thrombophilia
- Acquired thrombophilia testing is for all women with unprovoked VTE
- Dose of LMWH
 - Depends on weight of patient

Weight	Enoxaparin (mg)	Dalteparin (units)	Tinzaparin (Units)
< 50kg	20	2500	3500
50-90 kg	40	5000	4500
91-130 kg	60	7500	7000
131-170 kg	80	10,000	9000
>170 kg	0.6 mg/kg/day	75 U/kg/day	75 U/kg/day

- High prophylactic dose for women with weight 50-90 kg

Enoxaparin – 40mg BD

Dalteparin – 5000 U BD

Tinzaparin – 4500 U BD

Contraindication to LMWH: 00:56:43

1. Known bleeding disorder
2. Active antenatal/postpartum bleeding
3. Women considered at risk of major hemorrhage (e.g. placenta previa)
4. Thrombocytopenia (platelet count $<75,000/\text{mm}^3$)
5. Acute stroke in previous 4wks (Ischemia/Hemorrhagic)
6. Severe Renal disease
7. Severe liver disease

8. Uncontrolled hypertension

- Systolic BP > 200 mmHg
- Diastolic Bp > 120 mmHg

Warfarin

01:00:28

- Not safe during pregnancy
- Teratogenic drug
- If patient is on warfarin – prior to conception

↓

Change to LMWH as soon as pregnancy is confirmed.

[Ideally within 2wks of missed period
(or) before 6wks of pregnancy]

- In case of recurrent VTE/Mechanical valves: keep on warfarin (in 2nd trimester)
- Postpartum – start 5-7 days after delivery in the postpartum period.
- Breastfeeding – safe

LOGIN FOR SUCCESS

Warfarin Embryopathy

01:05:39

- Seen in 5% of Antenatal women, exposed between 6-12 weeks
 - Hypoplasia of nasal bridge
 - Congenital heart defects
 - Ventriculomegaly
 - Agenesis of corpus callosum
 - Stippled epiphyses

- Also causes
 - Abortions
 - Still birth
 - Neuro developmental problems
 - Hemorrhage in the mother (PPH), Baby (Neonatal Hemorrhage)

When to stop LMWH

01:08:44

- Q – Prophylactic dose of LMWH – stopped at least 12hr prior to regional Anesthesia
- Q – Therapeutic dose of LMWH – stopped at least 24hr prior to regional Anesthesia
 - LMWH should be stopped – if there is any vaginal bleeding /once labour begins.
- How to monitor
 - HIT (Heparin Induced thrombocytopenia,Fractures/Osteopenia)
 - UFH causes
- Monitored by checking platelet count.
 - Drug of choice – LMWH
- For LMWH
 - Prophylactic dose of LMWH – needs no monitoring
 - Dose of LMWH – should be reduced in women with renal impairment.
 - LMWH – safe during breastfeeding

Other drugs

- Danaparoid } Safe in pregnancy &
Fondaparinux } Breast feeding

But Not the Drug of choice
Not licensed yet

- Waiting period before giving regional anesthesia:
for Danaparoid -24Hrs
for Fondaparinux -36-42Hrs
- Aspirin □ not recommended for Thromboprophylaxis

TUTORIALS

LOGIN FOR SUCCESS

ANATOMY OF FEMALE GENITAL TRACT- I

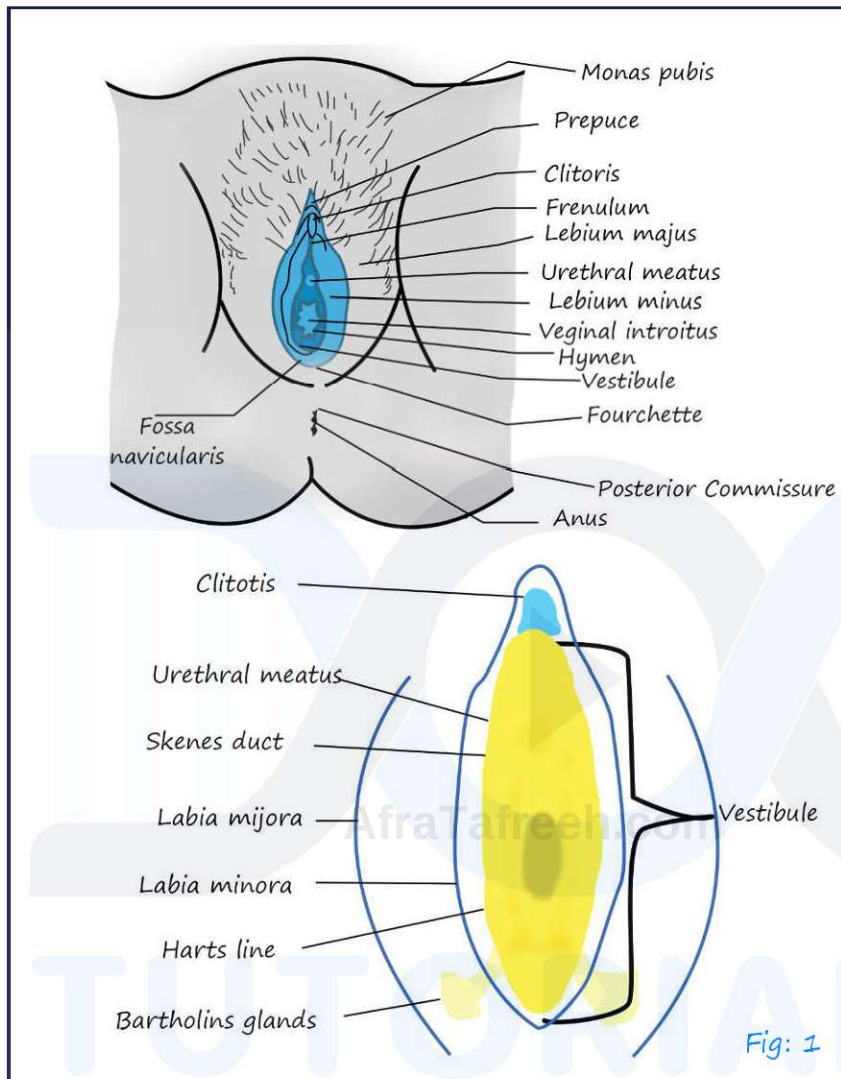


Fig: 1

Structures Include

- Mons Pubis
- Labia Majora
- Labia Minora
- Clitoris
- Hymen
- Vestibule
- Urethra opening
- Greater vestibular / Bartholin glands

- Minor Vestibular glands
- Paraurethral / Skene gland
- Mons pubis is thick pad of fat covered with hair (Sexual Hair)
- Hormone testosterone is needed for development of pubic / sexual Hair

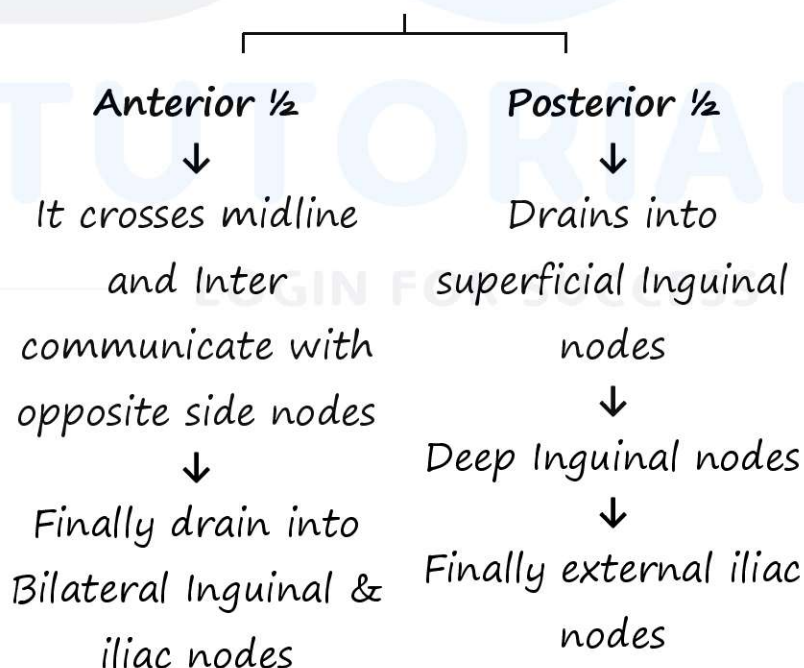
Homology of sexual organs

Female	Male
i. Ovary	Testis
ii. Para oophoron	Ductus deferens
iii. Paraurethral / Skene gland	Prostate
iv. Vagina (lower end)	Prostatic utricle
v. Clitoris	Penis
vi. Labia Majora	Scrotum
vii. Labia Minora	Urethral surface of penis
viii. Vestibular bulb	Corpus Spongiosum

Labia Majora

- It has thick pad of fat without muscle fiber. It is covered with skin having sexual hair
- 1. Homologous to scrotum in males
- 2. Covered with sexual hair
- 3. Skin :- Stratified squamous keratinized epithelium
- 4. It has no muscle fibers
 - It has only fat and elastic fibers
- 5. Both sebaceous glands and sweat glands present
- 6. Rich in lymphatic and venous plexus
- 7. Lymphatics of labia majora

Lymphatics of labia majora



- 8. Round Ligament terminates into labia majora at its upper border

Labia Minora

00:10:42

Labia Minora

- This labia Minora in its upper portion divides into two, which enclose the glands including tip of clitoris, thin tissue fold medial to labia majora
 - i. Superiorly divides into 2
 - Upper – Prepuce
 - Lower – Frenulum
 - ii. Inferiorly joins to form **FOUCHETTE**
 - iii. Two different types of epithelium cover labia minora separated by **line of HART**
 - Medial to HART line – non-Keratinized squamous epithelium
 - Lateral to HART line – Keratinized squamous epithelium
 - iv. It is devoid of sebaceous & sweat glands

Clitoris

00:13.02

Clitoris

- i. Organ of orgasm & homologous to penis in male
- ii. Located between prepuce and frenulum,
- iii. Tip is called **Glans**
- iv. Normal length – should not exceed 2 cm, if $> 2\text{cm}$, it is called **clitoromegaly**, which is a sign of **Hyperandrogenism**
- v. Length of glans – 0.5 cm or less
- vi. Arterial supply – deep and dorsal artery of clitoris

vii. Lymphatics – directly drain into Deep Inguinal lymph nodes

viii. Venous – Rich in plexus, drain into dorsal vein of clitoris

Vestibule

00:14:15

Vestibule-

- area enclosing all the openings in vulva

Boundaries

- Laterally – Hart line
- Medial – Hymen
- Anterior – Frenulum
- Posterior – Fourchette

Six openings

- Urethra
- Vagina
- 2 Bartholin glands
- 2 Skene / Paraurethral gland
 - Fossa Navicularis – Tissue between Hymen and Fourchette

Bartholin's Glands

00:17:37

Bartholin Glands

- i. Also called – Greater Vestibular gland
- ii. Gland size – 0.5 to 1 cm
- iii. Size of duct – 1.5 to 2 cm
- iv. Opening of Duct – 5'o and 7'o clock position

Para-urethral Glands

00:18:14

Para urethral Glands

- Also known as **Skene Glands**
- Paired glands – one pair on right, one pair on left along the inferior aspect of urethra
- Inflammation of glands –obstruction of ducts–
Urethral Diverticulum

INTERNAL GENETALIA

00:19:12

VAGINA

- i. Tubular structure extending from cervix to Hymenal opening.
 - ii. It has anterior wall, long posterior walls and 2 lateral walls.
 - iii. With Ectocervix, it forms fornices – anterior, posterior and 2 lateral fornices.
- Posterior fornix is deeper due to long posterior vaginal wall.

- iv. Epithelium: Non-Keratinized stratified squamous epithelium.
- v. Externally vagina opens at Hymen, which is membranous structure with small opening to allow drainage of menstrual blood & fluid
- vi. Due to repeated intercourse or during process of childbirth, Hymen ruptures and it is replaced by Rudimentary structure –**Myrtiform Caruncles**.
- vii. Boundaries:

- Anterior: Separated from bladder by uterovesical fold (uv fold) of peritoneum.
- Posterior: Separated from rectum by Rectovaginal fold of Peritoneum.
- Lateral wall: Up 1/8 – levator ani muscle

Length

- Anterior wall – 6 to 8 cm
- Posterior wall – 7 to 10 cm
- It has mucosal folds called Rugae or Vaginal Rugosities
- It has no glands
- It has smooth muscle, collagen and elastin

Blood Supply

- Upper vagina – Descending cervical / vaginal branch of uterine artery
- Posterior vagina – Middle rectal artery

- Distal part of Vagina – Internal pudendal artery

Lymphatic Drainage

- Upper 1/3 – Internal iliac, external iliac and common iliac nodes
- Middle 1/3 – Internal iliac Nodes
- Lower 1/3 – Inguinal lymph nodes

Uterus

00:25:22

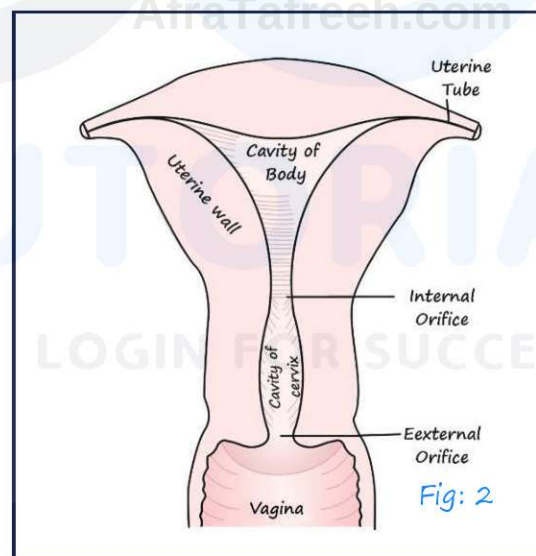
UTERUS

- Main female reproductive organ presents between Bladder anteriorly and rectum posteriorly
- Pear shaped organ.
- Uterus – Body or corpus and Cervix
- Length – Nullipara – 6 to 8 cm
 - Multipara – 9 to 10 cm
 - Weight of uterus – 60gms (non-pregnant stage)

CERVIX

- Lower part of uterus
- Lower most part of cervix protruding into vagina – Ectocervix, covered by external OS
- Cervix – Supra vaginal / Endocervix with internal OS
 - Infra vaginal / Ectocervix with external OS
- External OS corresponds to Ischial spines
- Internal OS corresponds to UV fold of peritoneum

- vi. Shape of Cervix - Before delivery → Oval / Round
After delivery → Transverse slit
- vii. Epithelium - Ectocervix - Stratified Squamous
- Endocervix - Columnar epithelium
- viii. Junction of both Ectocervix and Endocervix
corresponds to **Transformation Zone**, which is
more prone to ca of cervix
- ix. Eversion of Cervix - when columnar epithelium of
Endocervix exposes to vaginal PH. It is eversion of
Cervix
- x. It has little smooth muscle, mainly consist of
collagen and Elastic Fibrils, which helps to stretch
cervix during vaginal birth



Ligaments of Uterus

- i. Uterosacral ligaments
- ii. Mackenrodt's / Cardinal / Transverse cervical
ligament - **Main support of Uterus**

- iii. Round ligament
 - iv. Broad ligament
 - v. Ovarian ligament
- } **No role in support of Uterus**

Blood supply to round ligament → SAMPSON'S artery^Q

Blood Supply of Uterus

- i. Uterine Artery (Major part)
Ovarian Artery (Minor Part)
- ii. Ovarian Artery → Branch of internal iliac artery
→ Supplies mainly fundus of uterus and part of fallopian tubes.

Uterine Artery

- Branches into segmental / Arcuate artery

↓
Radial Artery

↓
Straight artery / Basal Artery → Supplies Basalis layer

↓
Spiral Artery → Supply functional layer of uterus

- Functional layer of uterus – Superficial compact layer
- Intermediate spongy layer

Lymphatics of Uterus and Cervix

Uterus

- i. Internal iliac nodes - **Mainly**
- ii. Para aortic nodes

Cervix

- Internal iliac Nodes

Nerve Supply

- Sympathetic - T₁₀ to L₂
 - Parasympathetic - S₂, S₃, S₄
- } Forms inferior hypogastric Plexus
- Inferior hypogastric plexus divides into
 - i. Vesical Plexus
 - ii. Middle Rectal plexus
 - iii. Utero Vaginal plexus
 - Pain from uterus - T₁₀ to L₂
 - Pain from Cervix, upper vaginal - S₂, S₃, S₄ (Pelvic splanchnic nerves)
 - Pain from lower vagina - Pudendal Nerve (S₂, S₃, S₄)
 - Pain fibers during uterine contractions carried by sympathetic T₁₀ to L₁

Ovaries

00:37.:25

Ovaries

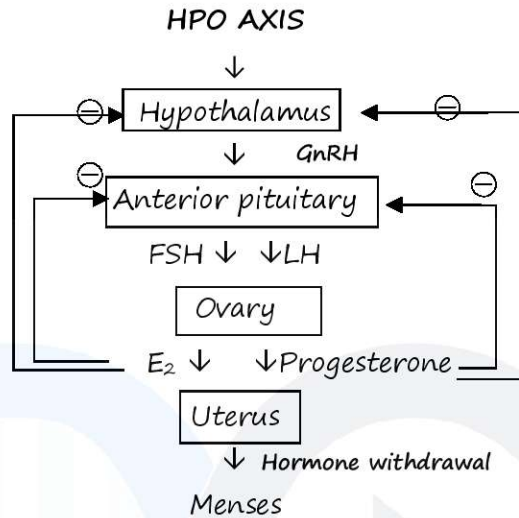
1. Paired, pearly white almond shaped structures
2. Dimensions – 3 x 2 x 1 cm
3. Ovary – Outer cortex – with follicles
Inner medulla – Blood vessels
4. Epithelium – Single layer of cuboidal epithelium
5. Tunica Albuginea → Connective tissue
surrounding the ovarian epithelium
6. Blood Supply – Mainly ovarian artery (90%) which
is anterior branch of internal iliac artery
7. Uterine artery (10%) (Ovarian ligament)
8. Nerve Supply – Sympathetic – T₁₀
9. Parasympathetic – Vagus nerve
10. Ovaries are homologous to testis in males developed
from coelomic epithelium embryologically.

LOGIN FOR SUCCESS

PHYSIOLOGY OF MENSTRUATION

Hypothalamus

00:00:16



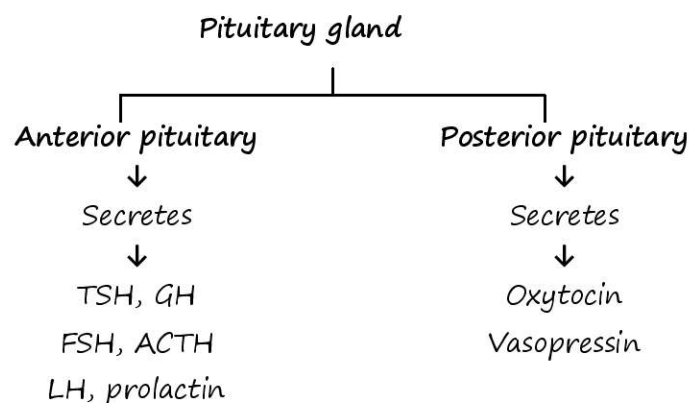
Hypothalamus

- Located at the base of brain
- Secretes
 - Gonadotropin releasing hormone (GnRH)
 - Thyrotropin releasing hormone (TRH)
 - Corticotropin releasing hormone (CRH)
 - Growth hormone releasing hormone
 - Dopamine

Pituitary Gland

00:00:38

Pituitary gland



GnRH

- Peptide hormone
- Secreted by hypothalamus
- $T_{1/2} \rightarrow 2 \text{ to } 4 \text{ minutes}$
- It Stimulates $\rightarrow$ Pituitary -release FSH, LH
- Released in pulsatile fashion
 - In follicular phase $\rightarrow$ Once in 60min
 - In luteal phase $\rightarrow$ Once in 90-120min

FSH

- Glycoprotein
- Stimulated by GnRH
- Inhibited by
 - Oestrogen
 - Inhibin
- Peaks on $\rightarrow D_6, D_{12}$
- Acts on granulosa cell
 - Helps in multiplication of granulosa cells
 - Increase LH receptors on theca cells
 - Increase aromatase activity (Peripheral oestrogen levels increase)

Luteinizing Hormone (LH)

- Glycoprotein
- Stimulated by $\rightarrow$ GnRH, high levels of estrogen
- Inhibited by $\rightarrow$ Low levels of estrogen
- Peaks 10-12hours prior to ovulation

Functions

- Final maturation of follicle & triggering ovulation
- Increase androgen production in theca cells
- Increase progesterone production in corpus luteum

Prolactin

- Polypeptide
- Inhibited by dopamine
- Stimulated by → TRH (Thyrotropin releasing hormone)
 - Vasopressin
 - VIP
- In hypothyroidism, as TRH increase - Prolactin increases

Stages of Follicular Development

00:08:46

Stages of follicular development

- **Primordial follicle:** Oocyte covered by single layer of flattened granulosa cells
- **Primary follicle:** Oocyte covered by multiple layers of granulosa
- **Secondary follicle:** oocyte covered by multiple layer of granulosa and theca cells, theca interna and externa
- **Tertiary follicle:** Characterised by antrum – which is fluid filled cavity

Ovulation

- Triggered by LH surge

Types of Estrogens

00:12:00

Types of oestrogens

	Source	Other name	Source	Potency
Estradiol	Ovary (Granulosa cell)	E_2	Testosterone	Most potent
Estrone	Adipose tissue	E_1	Androstenedione	
Estriol	Placenta	E_3		Least potent

- Normal duration of menstrual cycle $\rightarrow 28 \pm 7 \rightarrow 21-35$ days
- S. Estradiol on $D_2 \rightarrow 20-60$ pg./ml
- S. Estradiol prior to ovulation $\rightarrow 200-300$ pg/ml/Follicle
- Time gap between LH surge & ovulation $\rightarrow 36$ hours
- Time gap between LH peak & ovulation $\rightarrow 10-12$ hours
- S. progesterone before ovulation $\rightarrow <4$ ng/ml
- S. progesterone after ovulation $\rightarrow 4-40$ ng/ml

- Peak, activity of corpus luteum → 8 days post ovulation
- Maximum life span of corpus luteum → 14 days
 - Puberty: Time of onset of sexual menstruation
 - Gonadarche: Gonads start responding to FSH/LH
 - Menarche: Onset of menstruation
 - Thelarche: Onset of breast budding
 - Adrenarche: Onset of production of androgens by adrenal cortex
 - Pubarche: Onset of appearance of pubic hair

Puberty

00:19:00

Order of puberty development

- Growth spurt → Thelarche → Pubarche (Adrenarche) → Menarche
 - 1st sign of puberty → Growth spurt
 - 1st visible sign of → Thelarche
- **Delayed puberty:** Absent secondary sexual characters by 14 years
- **Precocious puberty:** Onset of secondary sexual characters by 8 years (or) Onset of menses before 10 years

PRECOCIOUS PUBERTY

Precocious Puberty

00:00:22

- Definition
 - Onset of secondary sexual changes/Pubertal changes before 8 yr.
 - (or)
 - Onset of menses before 10yr
- Types
 - i. Central
 - ii. Peripheral

Central Precocious puberty:	Peripheral precocious puberty
• Depends on GnRH • Causes: - Idiopathic → most common - Intracranial tumors/cysts - Infection - Trauma - Malformations - Gene mutations	• GnRH independent • Causes: - Estrogen secreting ovarian tumors - Sex-hormone secreting adrenal gland. - Mc-cune Albright syndrome - Exogenous estrogen exposure - Primary hypothyroidism

Examination

- Height
- Weight
- Skin lesions → café-au-lait spots seen in McCune-Albright syndrome
- Bony deformity
- Breast pubic & axillary hair –by Tanners staging
- Abdomen/Pelvic mass.

Investigation:

- Bone age
- Serum FSH, LH, TSH, Estradiol
- Ultrasound scan to rule out ovarian/adrenal mass
- MRI → to rule out Intracranial mass.
- Other hormone levels when indicated.

Treatment:

00:04:53

- Most effective drug → In treating central precocious puberty which is idiopathic → GnRH analogue

E.g.: Leuprolide

Peripheral precocious puberty:

- Surgery for main source of estrogen.
- Aromatase inhibitors → letrozole
Anastrozole
- SERM → Tamoxifen

DYSMENORRHEA

Dysmenorrhea

00:00:11

- Painful menstruation
- Incidence of dysmenorrhea – 50-70%

Difference between primary and secondary dysmenorrhea	
Primary (Spasmodic)	Secondary (Congestive)
Young/teens/before 20	> 20 yrs
Crampy/Recurrent pain	-
Mainly Suprapubic area	-
Radiates to back & thigh	-
Seen in regular ovulatory cycles	Regular/Irregular
Thin built girls	Obese
Associated with Nausea/vomiting	Associated with heaviness
Not associated with pelvic pathology	Pelvic pathology present
Begins with menstruation	Begins before menstruation (luteal phase) 1-2wks before onset of bleeding

Primary Dysmenorrhea

00:02:40

Risk Factors:

- Age <20 years
- BMI <20
- Nulliparity
- Early Menarche
- Heavy menstrual flow
- Anxiety/stress

Pathology: Progesterone – main hormone in 2nd phase of cycle

Ovulatory cycles



Increases progesterone



Increases Phospholipase A₂



Increases PG F_{2α}

Secondary dysmenorrhea

00:04:45

- Secondary dysmenorrhea

Causes are:

1. Fibroids

2. Endometriosis } Increases endometrial surface →
3. Adenomyosis } Increases PGF_{2α}

4. Pelvic inflammatory disease (due to congestion)

5. IUCD

6. Mullerian anomalies }
7. Cervical stenosis } Hematometra

8. Pelvic Congestion Syndrome

Treatment

00:06:35

- **Primary dysmenorrhea**

- NSAID → Decreases PG → Pain Decreases NSAID of choice
- Combined Oral Contraceptives (COC)
- Combined Transdermal Patches (E+P) Mefenamic acid
- DMPA (Depo-medroxy progesterone Acetate)

(or)

LNG – IUD (Levonorgestrel Intrauterine system)

→ Continuous progesterone

[COC – Combined Transdermal Patches] →

Anovulatory cycles



Decreases Progesterone

Decreases $PGF_{2\alpha}$



Pain Decreases

- Progesterone → Endogenous Estrogen Blocker
- Cyclical progesterone → D₁₅ – D₂₅ → Does not relieve dysmenorrhea
- Continuous progesterone → DMPA/LNG – IUD → relieves dysmenorrhea

Surgical Management

- TENS – Transcutaneous Electric Nerve Stimulation
- LUNA – Laparoscopic Uterine Nerve Ablation
- LPSN – Laparoscopic Presacral Neurectomy (T_{11} - T_{12})
- **Secondary dysmenorrhea**
 - Treatment of secondary dysmenorrhea → Treat the respective cause

Miscellaneous

00:09:06

- Unilateral dysmenorrhea
- Ovarian dysmenorrhea/Right ovarian vein syndrome
- Mittelschmerz pain
- Pelvic congestion syndrome

1. Unilateral Dysmenorrhea -

causes Q. NEET 2018

- Ovarian dysmenorrhea
- Bi cornuate uterus
- Unilateral pelvic endometriosis
- Unilateral Fibroid
- Colonic/cecal spasm
- Unicornuate uterus
- Rt ovarian vein syndrome

2. Ovarian dysmenorrhea

- Right ovarian vein syndrome

- Right ovarian vein crosses ureter at right angle.

When Pelvic congestion present



Engorgement of vein



Pressure on ureter



Stasis/UTI/Pyelonephritis



Pain

3. Mittelschmerz pain → at Hypogastrium/Iliac fossa

- Pain due to ovulation, due to stretching of ovarian capsule
- Increases tension because of stretching of follicle
- Irritation of peritoneum by follicular fluid
- Contraction of Fallopian tube – during ovulation
 - Sometimes associated with bleeding – Known as **Ovular bleeding.**
 - Sometimes associated with Nausea/Vomiting

4. Pelvic Congestion Syndrome

Autonomic disturbance



Gross vascular congestion



Pelvic varicosities



Congestive dysmenorrhea

Diagnosis

- History – 2 dysmenorrhea/Menorrhagia & Dyspareunia
- Investigation – Doppler → IOC
CT/MRI Angiography

Treatment

- NSAIDS
- MPA (Medroxy progesterone Acetate) 50mg daily x 4 months
- Hysterectomy



PRE- MENSTRUAL SYNDROME

Definition

00:00:19

- PMS encompasses a vast array of psychological as well as physical symptoms that cause significant impairment in the individual during luteal phase of the menstrual cycle

Diagnostic Criteria for PMDD (pre-menstrual dysphoric disorder)

00:02:09

Psychological Symptoms:

- ≥ 1 of following must be present
- Marked affective lability
- Irritability/anger/interpersonal conflicts
- Depressed mood
- Feeling of hopelessness
- Anxiety, tension

Physical Symptoms:

- Decreased interest
- Difficulty in concentrating
- Easy fatiguability, low energy
- Increase/decrease in sleep
- Feeling of being overwhelmed
- Breast tenderness
- Muscle/joint pain

- Bloating /wt gain
- ≥ 5 symptoms, occur in most cycles during the week before onset of menses
- Improves within a few days after menses
- Diminish in the week post menses
- Decreases quality of life/significant distress/interfere with routine work/school, shouldnot be underlying psychiatric disorders & shouldnot be aggravated by drugs
- Symptoms
 - Begins $\rightarrow$ Luteal phase
 - Decrease $\rightarrow$ As menstruation begins
 - Disappear $\rightarrow$ Post menstruation

PMS

AfraTafreeh.com

DRSP – Daily Record of Severity Problem 00:08:32

- DRSP atleast for 2 cycles
- Continuous GnRH analogue



Completely inhibit pituitary function



↓Symptoms

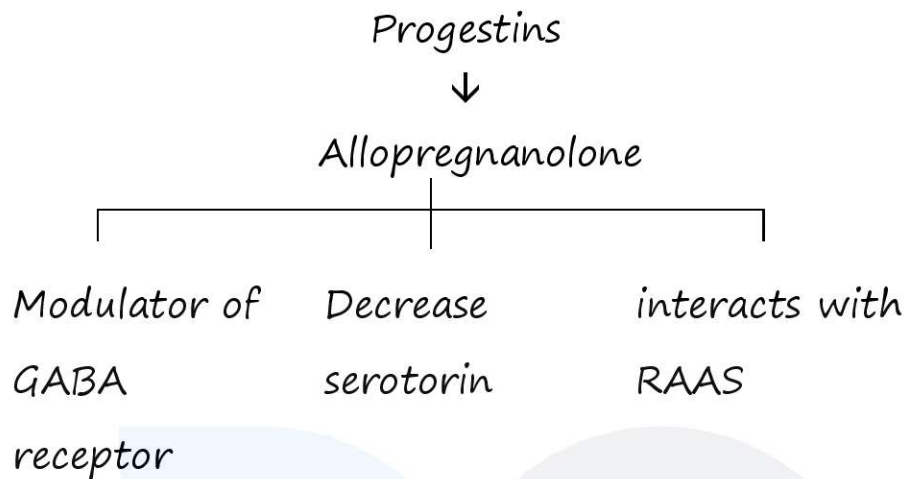
Pathophysiology

00:12:46

- Seen in ovulating cycle

Estrogen	E ₂ + Progesterone
	Luteal

- Estrogen – ↑ Neuronal excitability
- Progesterone – Neuronal inhibition



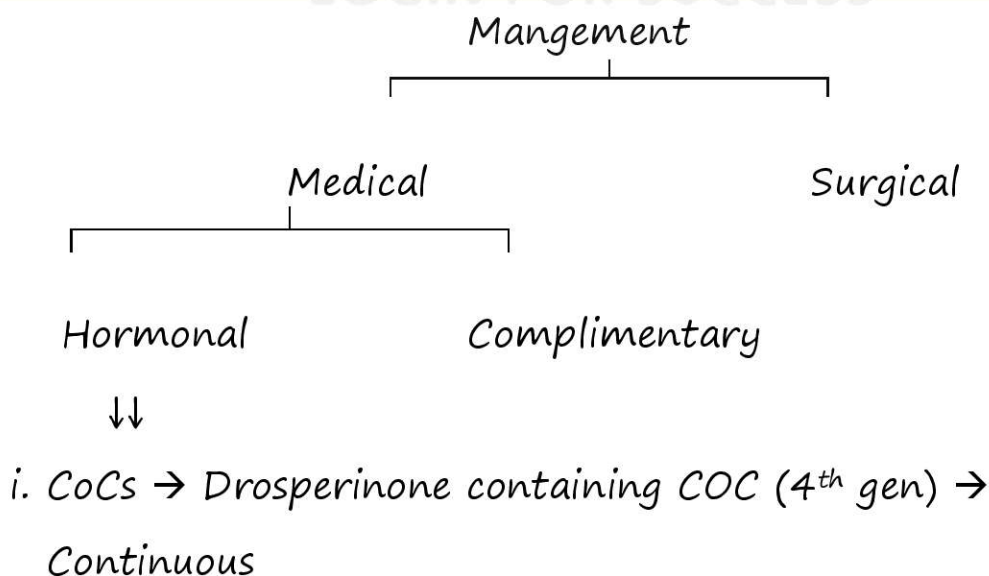
Variant PMD

00:16:44

- i. Premenstrual exacerbation of underlying disorder
 - EX: DM, Epilepsy, Asthma
- ii. Non – ovulatory PMD
- iii. Progesterone induced PMD

Management

00:18:12



ii. E_2 patch $\rightarrow$ 10-12 days; inhibits ovulation & cyclical progesterone

iii. E_2 patch & LNG IUS 52 $\rightarrow$ mirena $\rightarrow$ produces PMS like symptoms



Should not be used alone

iv. Danazol $\rightarrow$ androgen analogue; Virilization

- Inhibits Ovulation; relieves breast tenderness

v. GnRH analogues $\rightarrow$ only for severe most symptoms due to its side effects

- Maximum - 3 months: If $>$ 3 months

- Add back therapy with Continuous E + P (or) Tibolone

vi. SSRI/SNRI - 1st line; low dose - 10mg citalopram

- High dose - 20-40mg citalopram

-

vii. Spironolactone

• Other Drugs

i. Benzodiazepines

ii. NSAIDs

iii. Vit.E

Complimentary Therapy:

• Vit.B₆

• Exercise

• Calcium/Vit.D/Vit.E

- Magnesium
- Saffron
- Evening primrose oil (UFA)
- Ginkgo biloba
- Accupuncture
- Curcumin, wheat gain

Surgical Management:

- B/L oophorectomy is the surgery of choice

Decision Making Algorithm

00:35:52

-
- 1st line – low dose SSRI (continuous/luteal phase)
 - Exercise
 - CBT – Cognitive behavioural therapy
 - Vit.B₆, COC
 - 2nd line – Estrogen patch + micronized progesterone/
E₂ patch + Mirena
 - High dose SSRI
 - 3rd line – GnRH analogue + add back HRT
 - 4th line – B/L oophorectomy

GENITAL TUBERCULOSIS

- Genital TB is always secondary infection
- Most common primary site is lungs > Lymph nodes
- Most common site Involved – fallopian tube – Endosalpingitis
- M/C part affected in tube – Ampullary region
- M/C Age group is 20 to 30 years
- Organisms – Mycobacterium tuberculosis (m/c)
2nd m/c Mycobacterium bovis

Symptoms

- i. Mostly Genital TB – Asymptomatic
- ii. If Symptomatic, m/c symptom patients present with
 - Infertility.
- iii. Other symptoms
 - Menorrhagia
 - Hypomenorrhea
 - Amenorrhea
 - Associated with abdominal pain, low grade fever, loss of weight and Appetite
- iv. Parts involved
 - Fallopian Tubes (75– 100%) -Endometrium (30 – 80 %)
 - Ovaries (9 – 11 %)
- v. Tubal damage

- Partial occlusion of the tube leads to the - Ectopic pregnancy
- If tubes are Completely occluded - infertility
- vi. Pyometra is collection of pus in endometrial cavity.
- vii. Asherman syndrome - SCARS in endometrium.
- viii. Most common route of spread - hematogenous, since it is always a secondary infection from lungs. It can be sexually transmitted also - very rare

Diagnosis.

00:06:16

- i. Endometrial Biopsy - If suspected Endometrial involvement.
 - Unmarried Girls - Menstrual blood collection 12 hrs after menses onset ideally since we cannot take biopsy.
- ii. If suspected tubal involvement, Pus collected from tubes on laparoscopy, sample sent for
 - HPE
 - Culture in LJ medium
 - PCR (NAAT)
- iii. Gold standard technique - PCR (NAAT) from tube
- iv. Gold standard Diagnostic technique - Laparoscopy
Laparoscopy is done when patient presents with infertility or chronic PID or suspected tuboovarian mass

HYSTEROSALPINGOGRAM

not done in Active TB of genital tract, it can flare up the pelvic infection.

HSG Features of Tuberculosis.

00:07:23

- Lead pipe appearance of the tube
- Beaded appearance of the tube
- Hydrosalpinx
- Cornual block
- Intravasation of dye
- Golf stick tube
- Tobacco pouch appearance
- Honey comb appearance of uterus (also seen in Asherman's Syndrome)
- **Salpingitis Isthmica Nodosa** – also seen in Endometriosis

Treatment

- ATT (Anti tubercular drugs) for 6 months
- 2 HRZE + 4 HRE.

LOWER GENITAL INFECTION / VAGINAL INFECTION

- Infections of vagina are more common in Reproductive age group

Vaginal Anatomy

00:00:40

- Epithelium: Non-Keratinized stratified Squamous epithelium (Relatively resistant to infection)
- After puberty Estrogen acts on the vaginal epithelium



Increased glycogen deposition in cell



Attracts lactobacilli/ doderlein bacilli (commensal organism)

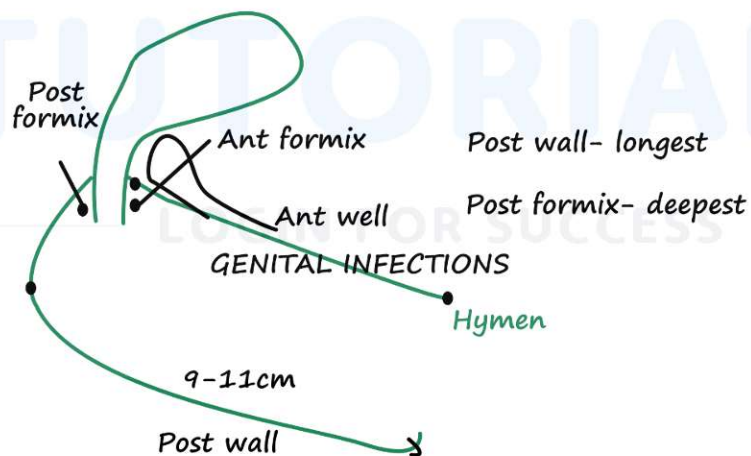
Convert Glycogen to Lactic acid (acidic PH of Vagina)

LOGIN FOR SUCCESS

Lactic acid protects from Infection

AGE	PH OF VAGINA
NEW BORN	3.5-4.5
6 WEEKS OLD CHILD	7
PUBERTY	Changes from ALKALINE to ACIDIC
REPRODUCTIVE AGE	4.5 - 5.5
PREGNANCY	3.5 - 4.5
POST MENOPAUSAL	>7

- During high oestrogen, so low ph levels.
- In new borns all the hormones present are derived from mother including oestrogen, so new born also has acidic vagina.



Etiology

00:07:06

- Trichomonas Vaginalis
- Bacterial Vaginosis
- Candidia
- Chlamydia

D.D of vaginal discharge

00:07:52

	Trichomonas	Bacterial vaginosis	Candidiasis	Chlamydia
i. Discharge	Thin greenish frothy	Thin greyish white	Curdy / Creamy	Purulent
ii. Causative organism	Parasite Trichomonas vaginalis - motile flagellated protozoan.	- Gardnerella vaginalis - Mobiluncus - Peptostreptococcus - Hemophilus (Polymicrobial)	Candida albicans	Chlamydia trachomatis
iii. Odour	Foul smelling	Foul smell (fishy odor)	No	Foul Smelling
iv. Mode of transmission	Sexual route/fomites	-	-	Sexually transmitted
v. Vag PH	>4.5	>4.5	<4.5	<4.5
vi. Pruritis	++++	-	++	+/-
vii. Diagnosis	Strawberry Vagina / Cervix - Wet mount	AMSEL'S Criteria	KOH mount	DNA PCR
viii. Treatment	Doc - Metronidazole	Metronidazole or clindamycin	AZOLE (fluconazole)	Azithromycin
ix. Partner treatment	Yes	-	-	Yes

- Max pruritus - trichomonas
- No pruritus - bacterial vaginosis.

- Criteria to diagnosis bacterial vaginosis
 - i. Thin greyish white discharge
 - ii. Vaginal PH > 4.5
 - iii. Presence of clue cells
 - iv. Whiff test positive – Fishy odour on adding NaOH / KOH to Vaginal discharge
- Clue cell – Large vaginal epithelial cells with bacteria present on their surface
- 3 out of 4 criteria required for diagnosis of bacterial vaginosis.

AfraTafreeh.com

TUTORIALS

LOGIN FOR SUCCESS

GENITAL INFECTIONS

Pelvic Inflammatory Disease (PID)

- i. Def: PID is the infection or inflammation of upper genital tract
- ii. Infection involving uterus,
 - Endometrium - Endometritis
 - Tubes - Salpingitis (Most common)
 - Ovaries - Oophoritis
- iii. Site most commonly involved in PID - Fallopian tubes

Risk Factors.

00:01:24

- Menstruating Teenagers
- Multiple sexual partners
- Pregnancy (immune compromised state)
- Menopause (Vaginal pH drops)
- IUCD (foreign body)

Protective Factors

00:01:42

- Barrier methods (condoms) and Hormonal contraceptives (OCP/POP) are protective
- Progesterone only pills or combined OC pills increases the thickness of cervical mucus and render protection against PID

Symptoms

00:02:43

- Patients usually present with
 - i. Lower Abdominal pain
 - ii. Fever with chills and rigors
 - iii. Tenderness in lower abdomen
 - iv. Abnormal vaginal discharge

Diagnostic Criteria of PID (CDC 2006). 00:03:11

i. Minimum Criteria	ii. Additional Criteria	iii. Definitive Criteria
a. Lower Abdominal tenderness	a. Oral temp > 38.3° C (fever)	a. HPE Evidence of Endometritis
b. Adnexal tenderness	b. Mucopurulent cervical / Vaginal discharge	b. Imaging by transvaginal Ultrasound/ MRI of Tubo ovarian Mass
c. Cervical motion tenderness	c. Raised ESR / CRP	c. Laproscopic Evidence of PID
	d. Lab documentation of infection with Gonorrhea or Chlamydia	Red swollen tubes with pus pouring out of fimbriae

For diagnosis of PID

Note: 1 minimum Criteria

+

1 Additional Criteria

+

1 Definitive Criteria

Indications for inpatient treatment (CDC 2006).

00:06:14

- i. Surgical emergencies like Appendicitis cannot be ruled out
- ii. Patient is pregnant
- iii. HIV positive (Immuno compromised)
- iv. Does not respond to oral antibiotics (within 72 hours)
- v. Intolerance to oral drugs
- vi. Severe illness – Nausea, vomiting, high fever
- vii. Patient has Tuboovarian (TO) abscess.

LOGIN FOR SUCCESS

Drug regimens

	OP Management	IP Management
Regimen A	i. Levofloxacin / Ofloxacin + Metronidazole	Cefoxitine + Doxycycline
Regimen B	Stat dose of ceftriaxone 250mg + Doxycycline 100 mg BD + Metronidazole 400 mg TID → For 7 to 14 days	IV clindamycin + IV Gentamycin

TUTORIALS

LOGIN FOR SUCCESS

FIBROID UTERUS -RISK FACTORS & TYPES

Fibroid uterus

- Also known as leiomyoma/myoma/fibromyoma.
- Most common benign tumor of the uterus.
- Most common solid tumor in a female.
- Contains smooth muscle cells and fibrous connective tissue.
- Site of origin is smooth muscle cells of myometrium.
- Incidence $\square$ 20% at the age of 30.
- Highest prevalence @ 35 years to 45 years

Etiology

00:02:55

-
- It is hormone dependent tumor (depends on estrogen & progesterone for its growth)
 - Depends on growth factors like Transforming Growth factor (TGF), Epidermal growth factor (EGF) & Insulin like growth factor (IGF)
 - Family history significant
 - Rearrangement/deletion of chromosome 6&7.

Risk factors:

Factors increasing the risk of fibroids	Factors decreasing the risk of fibroids
i. Nulli parity ii. Obesity iii. PCOS iv. Hyper estrogenic states v. black women vi. High fat diet vii. Family history	i. Multiparity ii. Menopause iii. Combined OC Pills (pseudo pregnancy drug) iv. Smoking (down regulates estrogen receptors)

Types of fibroids:

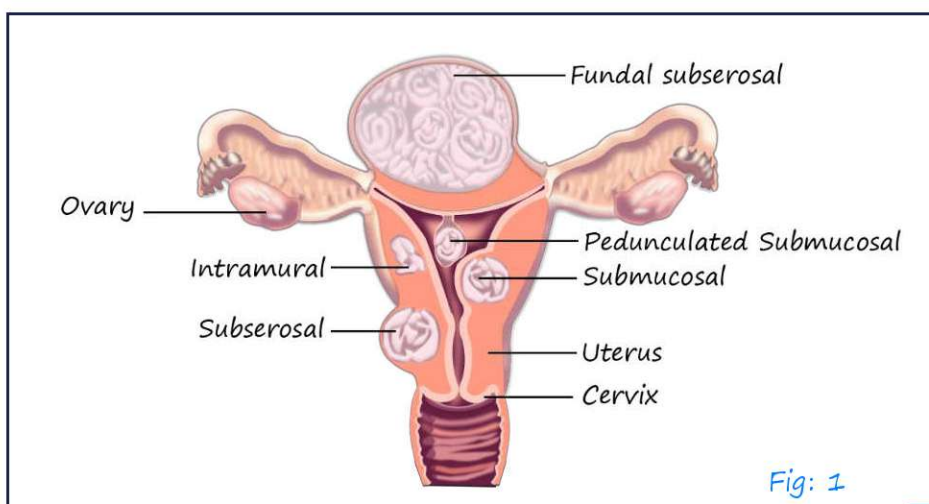
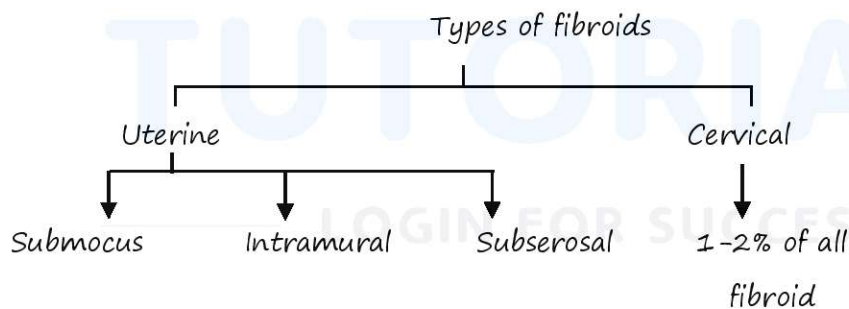
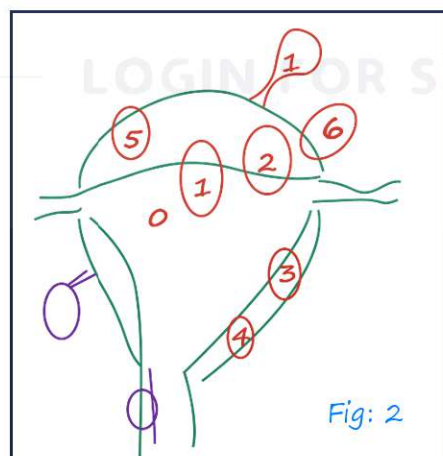


Fig: 1

FIGO classification of fibroids:

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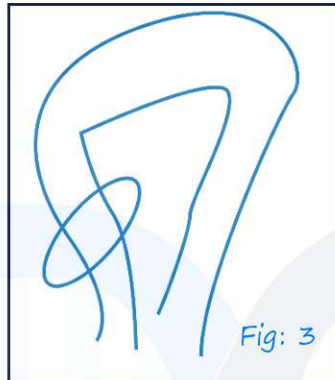
- 0 → Pedunculated submucous fibroid
- 1 → Partly submucosal and partly myometrium
<50% intramural (more into the cavity)
- 2 → Partly submucosal and partly myometrium
>50% intramural (more into myometrium)
- 3 → Completely intramural but touching the endometrium
- 4 → Completely intramural but not touching the endometrium
- 5 → Partly into myometrium & serosa (>50% in myometrium)
- 6 → Partly into myometrium & serosa (>50% in serosa)
- 7 → Completely pedunculated subserosal fibroid.
- 8 → Extra uterine fibroid □ cervix/broad ligament



- Submucosal → 0,1,2
- Others 3,4,5,6,7,8

Transmural fibroid-

- Extending into both uterine cavity and peritoneum
 - Hybrid fibroid
- 2 numbers are assigned to the fibroid.



- Measure the sub serosal, myometrial endometrial portions
- The first number represents the relationship of fibroid to the endometrial cavity, whereas, the second number represents that of the fibroid & peritoneal cavity.
- This fibroid number is 2-5.

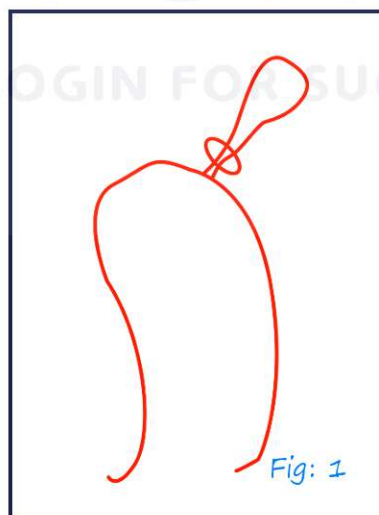
DEGENERATION

Intramural: (Most common 75%)

- Cell of origin – smooth muscle cell inside the myometrium
- All fibroid are initially intramural and then due to gradual contraction they can be pushed to endometrial cavity (or) peritoneal cavity there by becoming submucosal (or) sub serosal fibroid

Sub peritoneal / sub serosal fibroid:. 00:01:19

- They can be pedunculated / sessile
- If they are pedunculated there can be twist/ torsion of the pedunculated area and they can detach and get attached to surrounding structures and start growing known as **wandering fibroid**



- Wandering / parasitic fibroid is a feature of subserous fibroid / sub peritoneal fibroid

- With fibroids there can be ascites, pleural effusion (right > left)
- And this combination is seen with fibroid called **as pseudomeigs syndrome**
- Meigs syndrome: causally seen with ovarian tumor
- Peritoneal fibroid can also cause peritoneal hemorrhage

Sub mucus fibroid

00:03:07

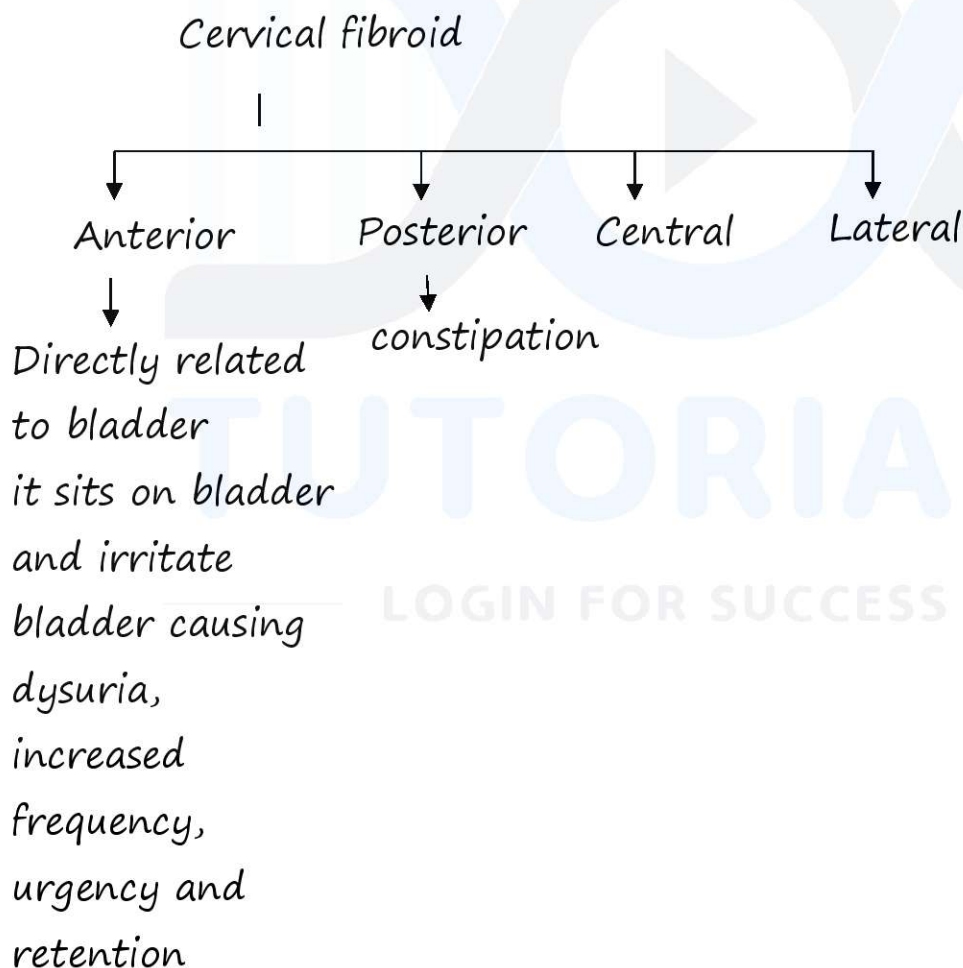
- Least common among uterine types
- Most symptomatic because it is in the endometrial cavity,
- It can grow and protrude through the cervical canal and vagina and can be called as fibroid polyp
- Most symptomatic because it has chances of undergoing necrosis, infection and can undergo malignant transformation because it is in direct contact with uterus cavity.
- Usually menorrhagia (non-cyclical bleeding) is seen in all types of fibroid, either subserous, intramural / party submucosal: but in case of sub mucus fibroid because there are chances of erosion & necrosis it can lead to metrorrhagia (a cyclical bleeding) (or) intermenstrual bleed
- And most vascular will be sub serosal and least sub vascular will be sub mucosal.

- Sarcomatous change is most common in sub mucous fibroid
- As you go from outside to inside vascularity gradually decreases.
- As least vascular one is more susceptible to degeneration and sarcomatous change.

Cervical fibroid

00:06:12

- Overall, least common



Central

- Grows to a huge size, uterus like sitting on a mass.
It is diffuse enlargement of the cervix on which uterus sits on the fibroid appearing as **lantern on dome of st. pauls church**.

Lateral fibroid

- Compress the ureter because ureter passes lateral to the cervix causing obstruction leading to **hydroureto nephrosis (HUN)**
- HUN can also be seen in broad ligament fibroid
- Broad ligament fibroid is not a true fibroid as itself moves from intramural site (or) subserosal site into the broad ligament causing **pseudofibroid**
- Broad ligament fibroid and cervical fibroid cause **hydro uretro nephrosis**

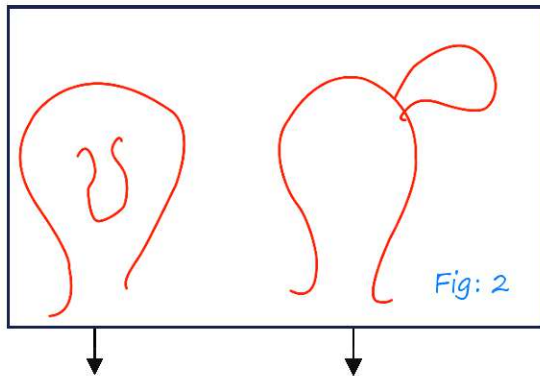
Pseudo cervical fibroid

- Is a fibroid which usually arises from the uterine cavity, which protrudes and distends the cervical canal
- On speculum examination it appears to be a cervical fibroid but it actually arises from the uterus

On examination

- Uterus is enlarged it can be symmetrical / asymmetrical enlargement if the fibroid occupies the

uterine cavity, it is a submucous fibroid and usually symmetrical

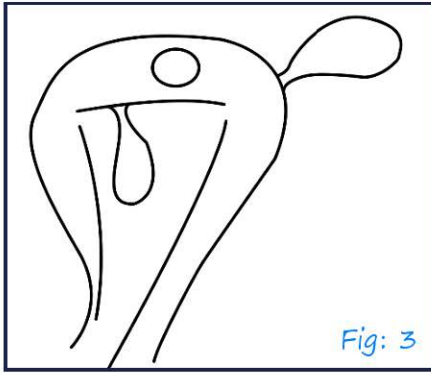


Symmetrical enlargement (sub mucosal) Asymmetrical enlargement (sub-serosal)

- Surface could be regular / irregular
- Firm in consistency
- Cut surface is smooth, whitish and whorled appearance/ trabeculation
- Whorled appearance due to fibrous component of fibroid
- Capsulated - a **false capsule**; due to compressed adjacent myometrium-form capsule

Secondary changes / Complications: 00:13:51

- Most commonly secondary changes are associated with blood supply
- Most vascular -sub serosal fibroid
- Least vascular -sub mucosal fibroid



- Sub mucosal fibroid
- Secondary changes are maximum and malignancy more common.
- In an individual fibroid of any type. Peripheral portion of fibroid receives more blood supply compared to central
- Degeneration of fibroid would actually begin from the central part fibroid and reach periphery

Secondary changes

00:16:16

1. Degenerative changes → sub mucous fibroid most commonly shows degenerative changes.
2. Atrophy - postmenopausal women and in puerperium
3. Necrosis
4. Infection
5. Vascular changes
6. Malignant changes
 - Necrosis & infection - most common in sub mucus fibroid

- Sarcomatous change → sub mucous because of least vascularity
- Sarcoma arising in the fibroid is leiomyosarcoma (LMS)
- Out of all leiomyosarcoma 0.3 to 0.8% of LMS arise from fibroid
- Out of all fibroid < 0.1% undergo malignant transformation

Degenerative changes

- Hyaline degeneration (most common)
- Cystic degeneration
- Fatty degeneration
- Red degeneration
- Calcification

Hyaline degeneration

- Centre to periphery is the usual pattern of spread
- Firm consistency of the fibroid is lost and it becomes soft and elastic
- Most common

Cystic Degeneration

- It is secondary to hyaline degeneration
- Hyaline degeneration followed by liquefaction of the degenerated areas leading to cystic spaces start appearing and are called cystic degeneration and is

usually seen after menopause and is **seen in intramural fibroids**

Fatty degeneration and Calcification

- Very rare, only 10% undergo calcific degeneration
- Calcium salts deposited here are: calcium carbonate & calcium phosphate.
- So, periphery is more vascular it spreads from periphery to the center because most vascular areas undergo calcification, calcium salts are derived from blood
- As subserous fibroid more vascular than any other fibroid, calcification is more in sub serous fibroid.
- Cystic degeneration – intramural fibroid
- Calcification – subserous fibroid
- And calcification is usually secondary to fatty degeneration
- Sometime the entire fibroid becomes calcified and become very hard known as **womb stone**

Red degeneration

- Other name: **carneous degeneration**
- Usually seen in large fibroid
- And it usually complicates 2nd half of pregnancy (late 2nd trimester and 3rd trimester)

Etiology of red degeneration

- Any hypercoagulable condition would lead to thrombus formation inside the myoma and block blood supply
- As pregnancy is a hypercoaguable condition, red degeneration is usually seen during pregnancy

Basic Pathology:

- Hypercoagulability → Thrombosis → Necrosis known as Necrobiosis
 - It is not caused by any infection; it is sterile
 - As there is necrosis, fibroid appears **Beefy red appearance / raw beef appearance**
 - It produces fishy odour

Management of red degeneration:

- NSAIDS / Pain killers & Rest
- No operative management required
- It is a self-limiting condition

SYMPTOMS & TREATMENT

Symptoms of Fibroid

- 75% asymptomatic
- MC symptom – Menorrhagia^Q
- 30% associated with endometriosis

Endometrial hyperplasia	} Causes for Menorrhagia
Increases surface area	
Venous Congestion	
Decrease in TXA ₂ : PGI ₂ ratio	

TXA₂ is a vasoconstrictor and PGI₂ is a vasodilator.

- Metrorrhagia in sub mucus fibroids
 - Infertility
 - Dysmenorrhea (congestive)^Q
 - Dyspareunia
 - Mass abdomen
 - Pain abdomen
 - Pressure symptoms → Retention of urine, constipation, hydrouretronephrosis
 - Anemia
 - **Polycythemia** – more common in broad ligment fibroi
 - Hydrouretro nephrosis → stasis in kidney → Increase erythropoietin levels
- Or
- Tumor itself secretes erythropoietin

Pregnancy Related Complications

00:05:40

- IUGR
- Preterm labour
- Cervical dystocia
- PPH
- Increases chances of C section
- PROM
- Malpresentation

Causes of Symmetrical Enlargement of Uterus

00:7:05

1. Pregnancy
2. Adenomyosis
3. Sub mucus/ intramural fibroid
4. Myohyperplasia
5. Pyometra
6. Hematometra
7. Lochiometra
8. Malignancies (endometrial Ca, Sarcoma)

On Examination

- Uterus can be enlarged to any size
- Regular/irregular, firm in consistency
- Lower border not felt
- Side to side mobility present but vertical mobility is absent

- Groove sign absent
- On bimanual pelvic examination a groove is felt in between the uterus and the mass if the mass is arising from the ovary because ovary is separate from uterus
- In Uterine mass, groove sign is absent
- In pedunculated sub-serous fibroid groove sign is present
- Transmitted mobility is present. (When you move the enlarged uterus, the movements are felt in the cervix)

Adnexal/Ovarian mass/ Sub serous fibroid	Fibroid
• Groove sign + • Mobility: transverse + Vertical + • Lower border –not felt • Transmitted mobility –	- Only side to side + - Vertical - mobility– absent - Lower border Not felt - transmitted mobility +

- IOC-USG
- <12 WKS –TVS
- >12WKS –TAS
- MRI → gold standard investigation

Management.

00:16:03

- i. Observation/Expectant
- ii. Medical
- iii. Surgical
- iv. Recent advances

Expectant Management:

- Asymptomatic fibroids
- Post-menopausal fibroids
- Fibroid in Pregnancy
- Fibroid in Puerperium

Medical Management → temporary solution

Mnemonic – good morning all delhi police

- Good – GnRH analogue (medical oophorectomy)
 - GnRH antagonist
 - Gestrinone
- Morning – Mirena – LNG IUD
 - Mifepristone – Progesterone receptor antagonist
- All – Asoprisnil/SPRM (ulipristil)
- Delhi – Danazol
- Police → Progesterone → only control bleeding
- For pain – NSAIDS

Indication for medical management

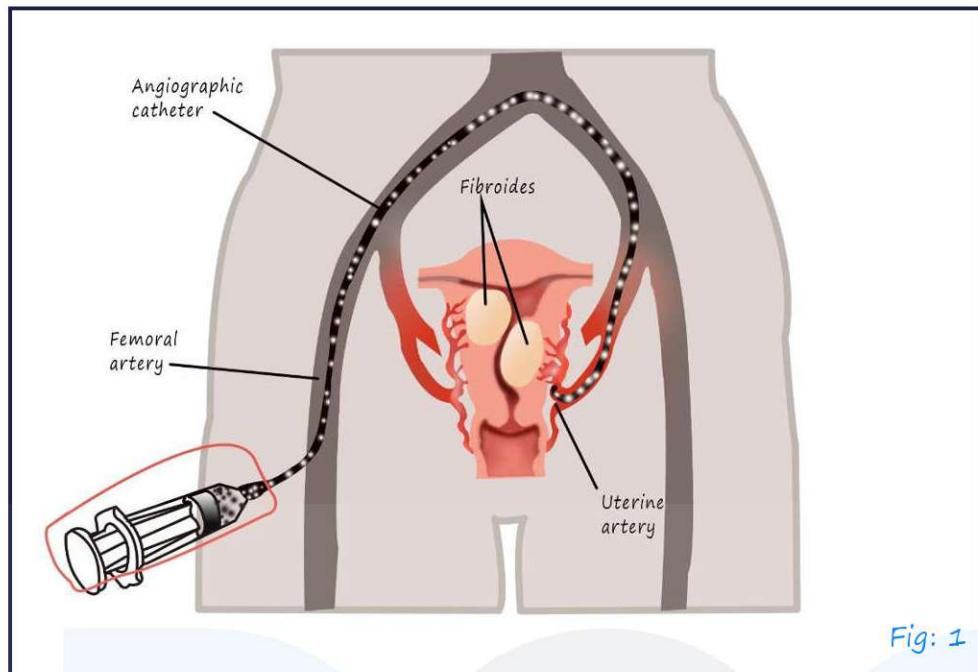
- Anemia
- Waiting period for social reasons
- Huge fibroids – To shrink fibroids – convert laparotomy to laparoscopy
- Decreases vascularity – treat her with preoperative gnrh



Recent Advances

Uterine artery embolization

- Vascularity of fibroid is compromised
- Inject polyvinyl alcohol (PVA) → occludes vessels



Complications

- Injury to femoral artery
- Permanently compromises reproductive function

Success Rate

- Menorrhagia improves (80-90% people)
- Shrinkage in 60% people

MRGF US

- Magnetic Resonance guided focussed ultrasound
- Also known as HIFU (high intensity focused ultrasound)
- Thermal ablation of fibroid occurs
- Thermal ablation
 - ↓
 - Coagulation necrosis

Advantages

- *Safe*
- *Easy*
- *Minimally invasive*

Disadvantages

- *Multiple setting*
- *Compromise future fertility*



ENDOMETRIOSIS & ADENOMYOSIS

Endometriosis

- Leading Cause of infertility
- Difficult to Treat
- Invasive, premalignant

Definition

- Presence of functioning endometrium in sites other than uterine mucosa.



Within myometrium

Also Known as

“Adenomyosis”

- Benign / Locally Invasive
- Disseminates Widely
- Prevalence rate 30%

True Endometriosis

Sites of Endometriosis

Common sites	Remote sites
Ovary → m/c site	Umbilicus
Pelvic peritoneum	Scar
Pouch of Douglas	Lungs
Utero Sacral Ligaments	Pleura
Recto – Vaginal septum	Ureter
Sigmoid colon	Kidney
Appendix	Arms / Legs
Lymph node	Nasal Mucosa
Fallopian Tube	

Risk factor of Endometriosis

00:07:14

- More exposure to Estrogen



Endometrial Growth

- Low parity
- Delayed child bearing
- Family history
- Imperforate hymen
- Environmental toxin – DIOXIN
- Molecular defects
 - Cytokines
 - Tumor Necrosis factors Alpha

- Macrophages
- Matrix Metalloproteinase
- Interleukins
- VEGF
- Estrogen

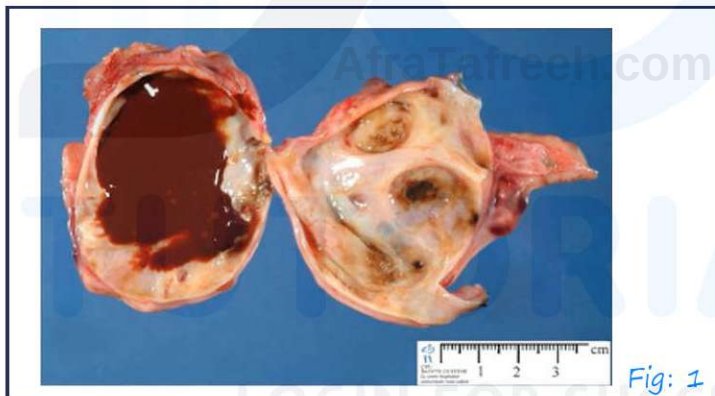
Theories

00:11:11

1. Retrograde menstruation theory (Sampson's theory)
2. Coelomic metaplasia (Mayer & Iranoff theory)
3. Direct implantation
4. Lymphatic theory (Halban's theory)
5. Vascular theory
6. Genetic/Immunological factor

Sites	Theory
Pelvic endometriosis	Retrograde menstruation Coelomic metaplasia
Abdominal viscera/ (Recto – Vaginal Septum) umbilicus	Coelomic metaplasia
Scar, vagina, cervix	Direct spread
Lymphnode	Lymphatic Spread
Distant site	Vascular

- Proliferative endometrium
 - ↓
 - Cyclical growth/shedding
 - ↓
 - Irritant blood
 - ↓
 - Adhesion / Puckering → Powder Burnt areas / Matchstick Spot
 - ↓
 - Encysted with cyclical bleeding
 - ↓
 - Chocolate cyst Brown – **Black in color**

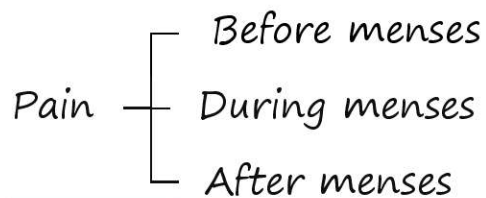


- On Usg → True Capsule over Chocolate cyst.
 - Thick walled, Homogenous cyst with Minimal internal echoes

Symptoms

- Most common: 30 – 45 years
- 25% → Asymptomatic

- Non-pigmented lesion are more Symptomatic than pigmented Lesion
- Most common
 - Dysmenorrhea
 - Congestive/Secondary
- Triple dysmenorrhea



Diagnosis

00:26:57

- Menorrhagia
- Premenstrual syndrome
- Infertility
- Dyspareunia
- Abdominal Pain
- Vicarious Menstruation (epistaxis during menses)

- Infertility → Due To

i. Scarring & puckering around ovaries & F.T



Relationship between ovary & FT is lost

ii. Ovarian wall – becomes thick

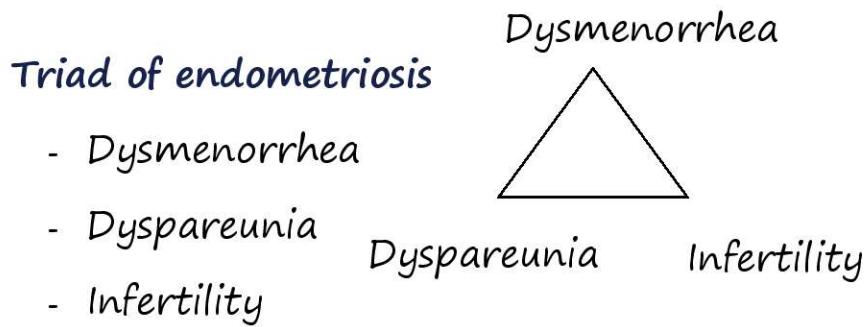


Ovulation does not occur

- Luteinised un ruptured follicle

- Vicarious Menstruation

- Epistaxis during menses (Due to endometriosis involving Nasal mucosa)

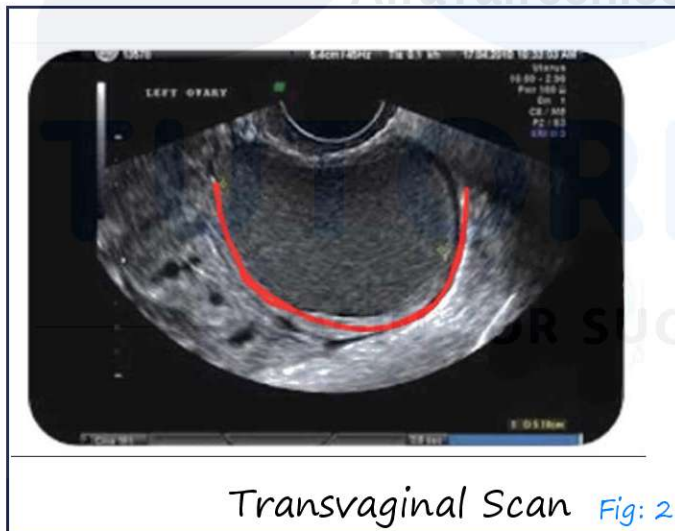


Serum markers

- Ca125 → Non specific
- MCP - 1 (Monocyte Chemotactic protein - 1)

USG Criteria To Diagnose

00:33:35



<i>Functional Cyst</i>	<i>Endometriotic Cyst</i>
4-5 Cm	>4-5 cm
Regresses with menses	Grows with every menstrual cycle
Clear	
Homogenously black	Internal Echos present
No internal Echos	Well capsulated

- Gold standard technique for diagnosing endometriosis
→ Laparoscopy

Endocrinopathy in Endometriosis

00:37:43

1. Corpus luteal insufficiency
2. LUF (Luteinised un ruptured follicle)
3. Hyper prolactinemia
4. Double LH peak

Staging: American Fertility Society Staging (AFS staging)

1. Size of Lesion

		<i>1cm</i>	<i>1-3 cm</i>	<i>3cm</i>
Peritoneum	<i>Superficial</i>	<i>1</i>	<i>2</i>	<i>4</i>
	<i>Deep</i>	<i>2</i>	<i>4</i>	<i>6</i>

Ovary	Right superficial	1	2	4
	Right deep	4	16	20
	Left superficial	1	2	4
	Left deep	4	16	20

2. Adhesions

		<1/3 enclosure	1/3 - 2/3	>2/3
Ovary	Right flimsy	1	2	4
	Right dense	4	8	16
	Left flimsy	1	2	4
	Left dense	4	8	16
Tubes	Right flimsy	1	2	4
	Left dense	4	8	16

3. Partial POD obliteration -4, complete -40

- Stage I – 1-5
- Stage II – 6-5
- Stage III 18-40
- Stage IV – >40 → Severe most Endometriosis

Medical Management

Drugs

00:44:55

1. Combined Oral Contraceptives
 - progesterone
2. Medroxy progesterone Acetate (MPA)
3. Dydrogestrone
4. Dienogest
5. Norethisterone
6. DMPA (Injectable)
7. Mirena (IUCD)
8. Danazol
9. Gestrone
10. GnRH analogue/antagonist
11. Mifepristone
12. SPRM (selective progesterone receptor modulator) –
Ulipristal, Asoprisnil
13. Anastrozole (Aromatase Inhibitor)
14. Simvastatin Antimitotic agents
15. Rosiglitazone

- Asymptomatic ^{Wait/expectant Treatment}
- Post menopause

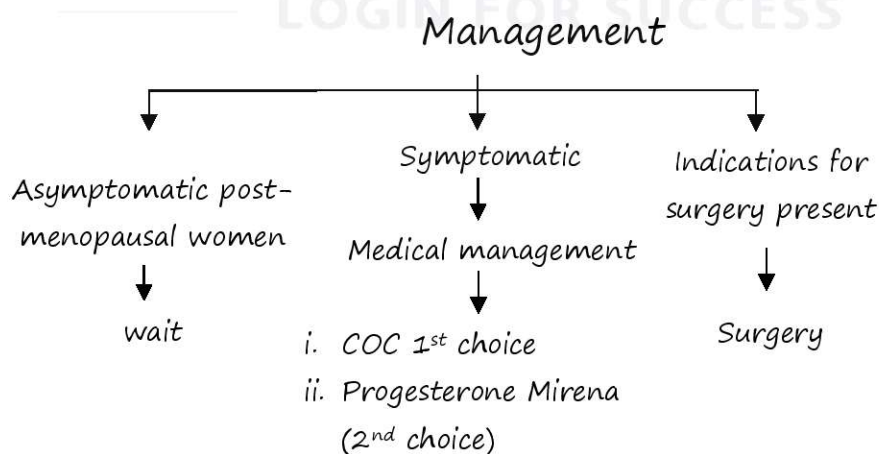
Indications of surgery

00:48:35

1. Not responding to medical Treatment
2. Deeply infiltrating lesions
3. Endometrium > 1cm

Types of surgery

1. Cauterisation
2. Laser vaporisation
3. LUNA (Laparoscopic uterine Nerve Ablation)
4. Adhesiolysis
5. Cyst excision $\geq 4\text{cm}$
Aspiration < 4cm
6. Laprotomy $\rightarrow$ Total Abdominal Hysterectomy with bilateral Salpingo oophorectomy



Adenomyosis

00:52:55

- K/A: Endometriosis Interna
 - Definition
 - Ingrowth of endometrial glands & stroma into the myometrium of uterus
 - Predisposing factors
 1. Repeated child birth
 2. Repeated dilatation & curettage
 3. Excess E_2
- } Disturb endomyometrial junction



Symptoms

00:57:36

- Hyperplasia of myometrium
- Diffuse enlargement of uterus (14 wks size)
 - No capsule
- Visible blood clots
- Cystic spaces within myometrium



- Hyperplasia of Myometrium
- True Capsule – Endometriosis
False capsule – fibroid
No capsule – Adenomyoma

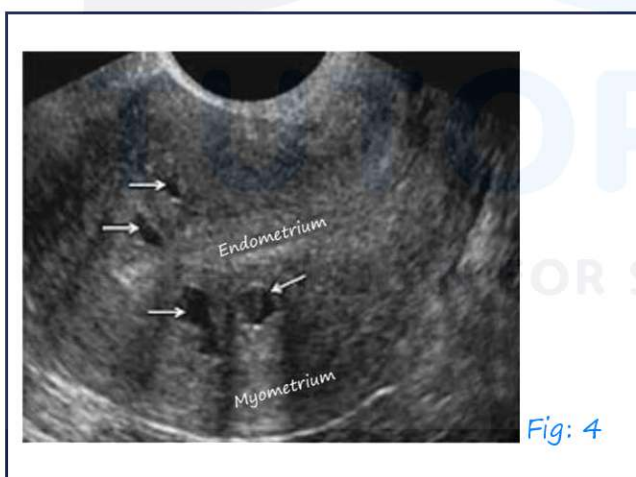
Diagnosis

- Menorrhagia 70%
- Dysmenorrhea 30%
- Dyspareunia
- Infertility
- Urinary disturbances

On USG

01:01:31

- Sub endometrial hypoechoic zone → Haze
- Heterogenous echogenicity with in myometrium (due to blood clot)
- Multiple cystic spaces
- Increase vascularity
- Ill-defined endometrial echoes



Investigation → Gold standard → MRI

- Junctional zone thickness → >12mm

Treatment

- For Pain → NSAIDS
- LNG IUCD / MIRENA } Not completed family

→ DANAZOL IUD

- *GOLD standard Treatment*

Hysterectomy / Adeno myomectomy in Completed family



ABNORMAL UTERINE BLEEDING PART- I

Old Nomenclature

00:00:32

- Amenorrhea – absent menses
- Oligo menorrhea – menses less frequent than every 35 days
- Polymenorrhea (epimenorrhoea) – menses more frequent than every 21 days
- Metrorrhagia – Menses at irregular intervals
- Menorrhagia (hypermenorrhoea) – menses lasting for >7 days or blood loss > 80ml

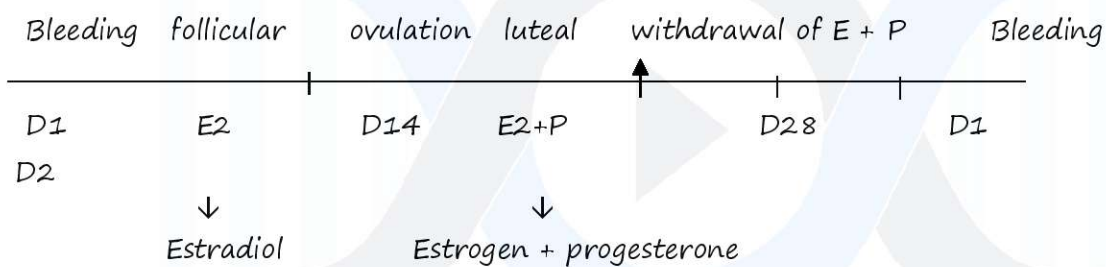
Current Descriptive Terms

Frequency	24-38 days	Frequent - < 24 days cycle interval Infrequent - > 38 days
Regularity	±2-20 days	Regular Irregular - > 20 days
Duration of flow	4-8 days	Shortened - < 4 days - hypomenorrhoea

		Prolonged - > 8 days - menorrhagia
Volume of blood loss	5-80ml	Light - < 5ml - hypomenorrhea Heavy - > 80ml - menorrhagia

Steroid Hormone signals in AUB

00:06:00



- Q ovulatory cycle = E+P withdrawal bleed Q

Mechanisms controlling onset of Menses

00:07:33

- E_2 produces PGE_2 & PGF_2 in 1:1 ratio
- Progesterone produces PGE_2 & PGF_2 in 1:2 ratio -
cause Vasoconstriction & control of bleeding
Progesterone acts only on estrogen primed
endometrium

Regression of corpus luteum



Fall in estrogen & progesterone



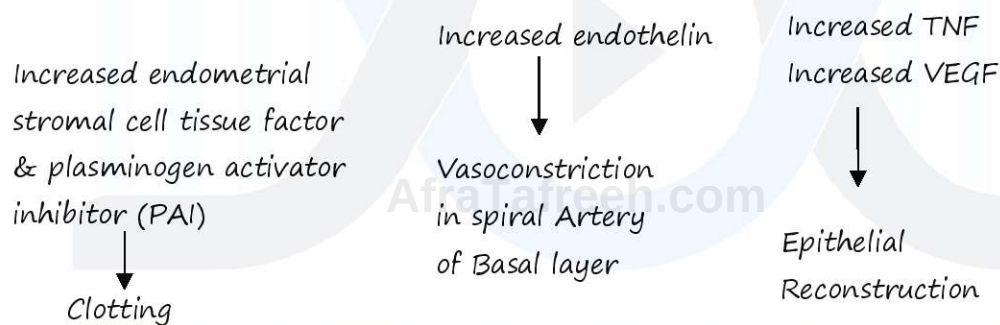
Increased chemokines IL₈, MCP-1, MMP



Bleeding begins

Mechanism controlling cessation of bleeding

00:10:27



Other types

- Estrogen withdrawal bleeding
- Estrogen break through bleeding
- Progesterone withdrawal bleeding
- Progesterone break through bleeding
- Break through bleed = fluctuating hormone levels – spotting seen

Estrogen withdrawal bleeding

00:14:00

- Due to complete absence of estrogen
- Due to sudden withdrawal of estrogen (ovary, peripheral tissue)

Examples:

- B/L oophorectomy in follicular phase
- Cyclical E_2 only HRT
- Preceding & following ovulation (ovular bleeding)

Estrogen break through bleeding

00:16:16

- Due to fluctuating levels of estrogen
- Seen in chronic anovulation ^Q **Ex: PCOS**

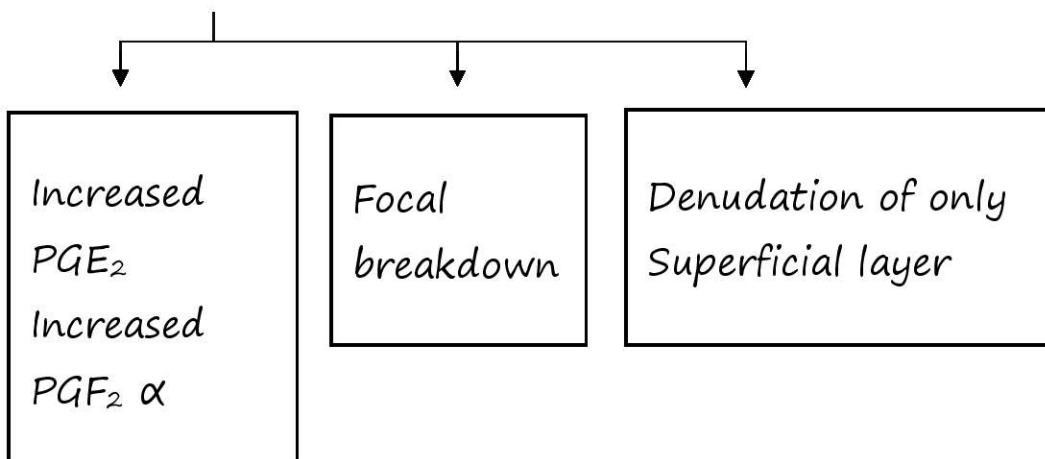


(Only estrogen, no progesterone)

- Changing cohort of follicles
(Fluctuant but uninterrupted E_2 levels)



Tissue outgrows its blood supply



Progesterone withdrawal bleeding 00:19:15

- Seen after discontinuing treatment with progesterone
 Q (cyclical progesterone therapy)

Progesterone break through bleeding 00:19:50

- Occurs with increased progesterone/estrogen ratio
- Seen with minipill (POP, implants, DMPA) Q
- Bleeding seen with COC (while on COC) Q
- On continues progesterone therapy

Differential diagnosis of AUB

PALM (structural)	COEIN (functional)
P – polyp	C – coagulopathy
A – Adenomyosis	O – Ovulatory dysfunction
L – Leiomyoma	E – Endometrial cause
M – Malignancy	I – Iatrogenic
	N – Not yet classified

Coagulopathy 00:24:56

- Von Willebrand's disease (MC)
- Glanzmann thrombasthenia
- Idiopathic thrombocytopenic purpura

Ovulating dysfunction

00:25:27

- All causes of anovulation
- Hypo/Hyperthyroidism
- PCOS
- Hyperprolactinemia

Endometrial dysfunction

00:25:56

- Acute endometritis Q
- Chronic endometritis
- Placental site nodule Q

Iatrogenic

00:26:20

- Hormonal contraceptives – COC/POP
- IUCD
- HRT
- Anticonvulsants, Anticoagulants
- Ginseng (estrogen herb)

LOGIN FOR SUCCESS

Not yet classified

- Chronic endometritis
- A-V malformation

Calculate Amount of Blood loss

00:27:30

NAME :		SCORE :							
DAY START :		DAY							
TOWEL		1	2	3	4	5	6	7	8
 1									
 5									
 20									
CLOTS/ FLOODING									
TAMPON		1	2	3	4	5	6	7	8
 1									
 5									
 10									
CLOTS/ FLOODING									

Fig: 1

- Small clot -1
- Large clot - 5

Total points > 100 = Menorrhagia

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Measuring Menstrual Blood Loss

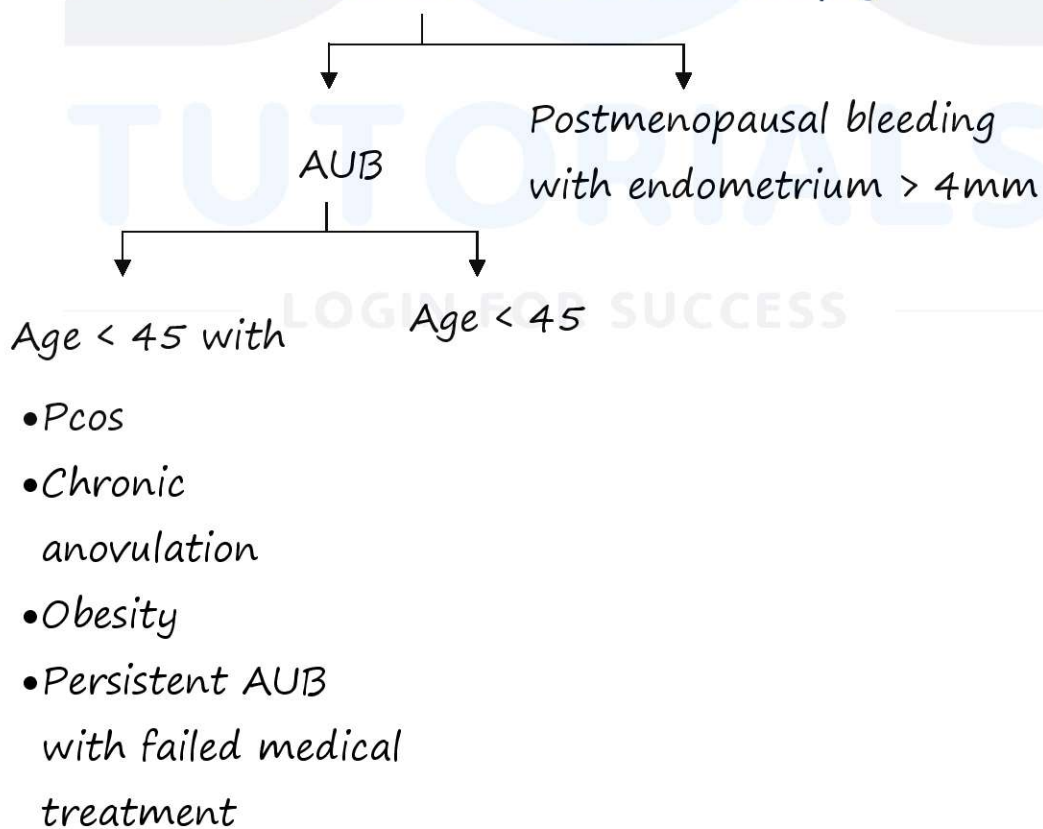
00:30:27

- Changing pads more than every 3 hours, more often
- > 20 pads/menses
- Need to change during night
- Frequent episodes of accidental soiling of clothes/bed sheets
- Passage of clots longer than one inch
- Menses lasting for > 7 days/Anemia

Investigations

Urine pregnancy test	FSH/E ₂ /AMH (for PCOS)
CBC	E. biopsy (for diagnosis of malignancy)
S progesterone	TVS (fibroid/polyp)
TSH/Prolactin	MRI/SSG
RFT/LFT	Hysteroscopy (overall best)
PT/ApTT	
PaP smear	

Indications for Endometrial Biopsy

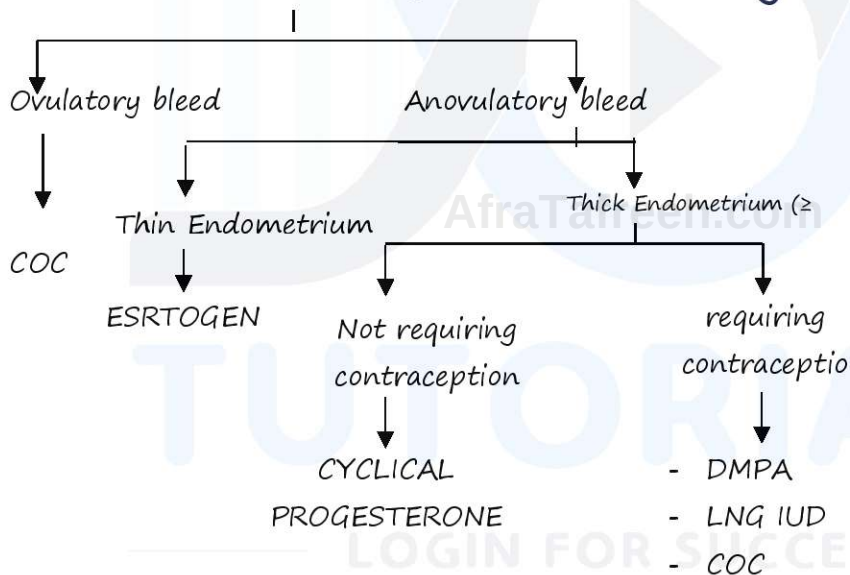


ABNORMAL UTERINE BLEEDING PART – II

Medical treatment for acute bleeding 00:00:30

- Tamponade
- Conjugated Equine estrogen either 25mg i.v every 4 hrs or 2.5mg every 6hrs (orally). Taper to once daily.
- Combined pills
- Oral medroxy progesterone acetate
- Tranexemic acid (IV or oral)

Medical treatment for chronic bleeding



• Other drugs used to control bleeding

- DMPA (Depot Medroxy Progesterone Acetate)
- NET-EN (Norethisterone Enanthate)
- Tranexemic acid
- NSAID
- Danazol
- GnRH agonist
- GnRH antagonist

Endometrial Ablation

- Reduces bleeding in 80-90% of women with heavy menstrual bleed.
- Amenorrhea occurs in 25-50% women, so women who want to retain menstrual cycles are not suitable candidates.

Contraindications for ablation

- Pregnancy
- Acute pelvic infection
- Endometrial hyperplasia (or) Genital malignancies
- Women wishing to preserve Uterus
- Post-menopausal women
- Expectation of amenorrhea
- Large or distorted endometrial cavity
- Intra uterine device in place
- Prior uterine surgery

Disadvantages of ablation

- i. Uterine synechiae → Hematometra (Distension and cyclical pain)
- ii. PATSS – Post ablation tubal sterilisation syndrome
- iii. Inadequate endometrial sampling post procedure
- iv. Difficult hysteroscopy or TVS evaluation

Generations of ablation

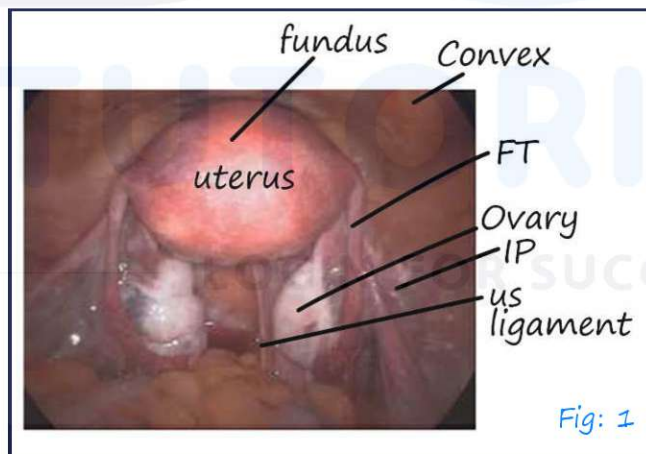
1 st generation	2 nd generation
- Hysteroscopy guided - Volume overload - Done under general anesthesia - For laser and bipolar techniques saline is used as distension media and for monopolar techniques glycine is used as distension media.	- Blind/not hysteroscopy guided - No volume overloads - i.v sedation/local anesthesia
• 1st generation technique includes: - Laser ablation (Nd Yag) - TCRE (Transcervical resection of Endometrium) - Roller ball coagulation	• 2nd generation techniques include: - Thermal ablation (81°C for 8 minutes) - Cryoablation (-100 to -120°C) - Novasure - Microwave ablation (75-80°C)

DEVELOPMENTAL ANOMALIES OF MULLERIAN DUCT

Mullerian Structures include.

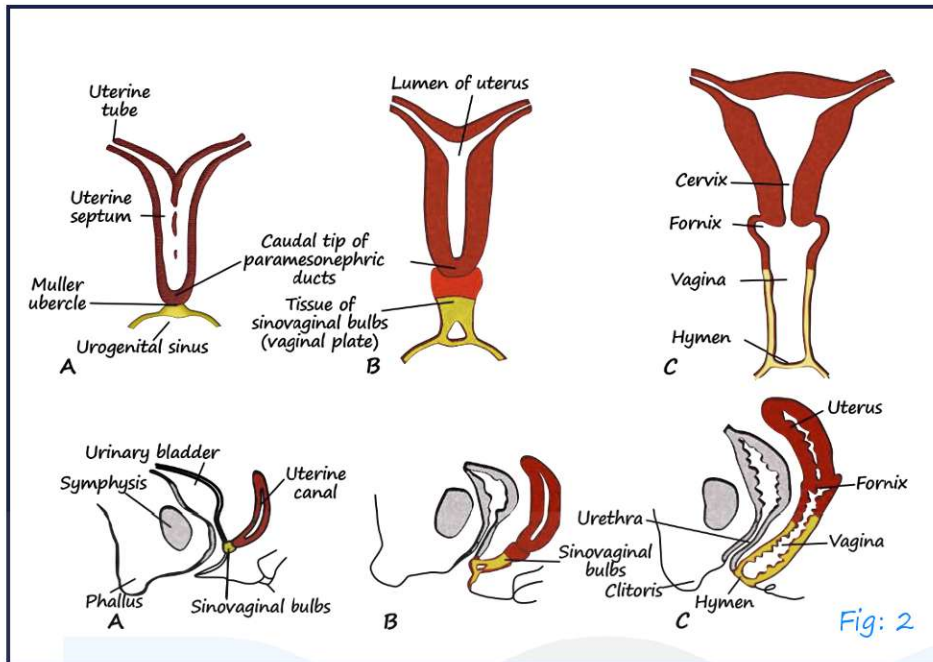
00:02:03

- Uterus, cervix
- Fallopian tubes
- Upper 2/3 of vagina
- They develop from paramesonephric duct/mullerian duct formed by invagination of coelomic epithelium
- The right and left mullerian duct unite in the midline to form uterus, cervix and upper part of vagina
- Proximal unfused part of mullerian duct forms Fallopian tubes



Paramesonephric Duct

- In the absence of testosterone and anti-Mullerian hormone, mullerian duct develops and wolffian duct regresses



Development of Vagina

- Vagina – Upper 2/3 → Mullerian Duct
- Lower 1/3 → Urogenital sinus
- Urogenital sinus forms with mullerian duct at a point called “Mullerian Tubercle”
- This Mullerian tubercle grows upwards to form Sinovaginal bulb or Vaginal plate

Sinovaginal bulb



Proliferation and Canalization of cells forms Vagina

- Cells in the Sinovaginal bulb degenerates and canalization occurs forming lower part of vagina

Fallopian tube

Uterus

duct

Cervix



Mullerian / Paramesonephric

Upper 2/3 of vagina

Lower 1/3 of vagina → Vaginal plate / Sinovaginal bulb (from Urogenital sinus)

Developmental Anomalies of Vagina. 00:06:44

i. Transverse vaginal septum -

Failure of canalization of vagina → Septum may be seen in upper part or lower part of vagina

ii. Imperforate Hymen:

Incidence 1 in 1000 to 1 in 2000

- Hymen -
- Partition between dilated canalized and fused sinovaginal bulb and urogenital sinus
- Hymen perforates shortly after or before birth
- Failure of perforation of Hymen leads to imperforate Hymen, due to which menstrual blood, cervical secretions get pooled up in uterine cavity
- Clinically, patient has Amenorrhea with suprapubic bulge and urinary retention
- Treatment is cruciate incision at base of Hymenal ring

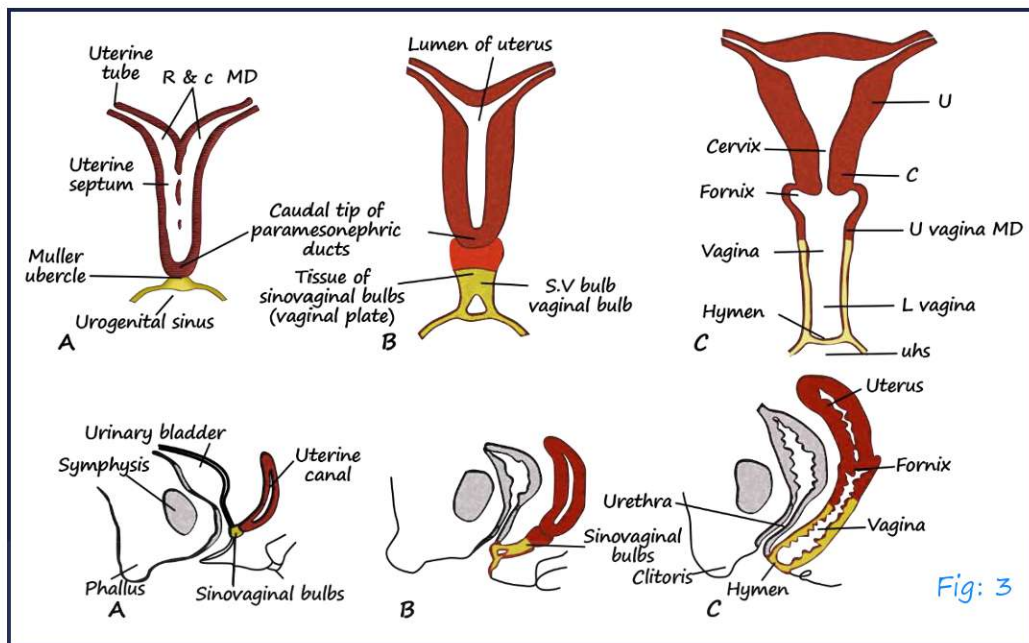


Fig: 3

Types of Hymens

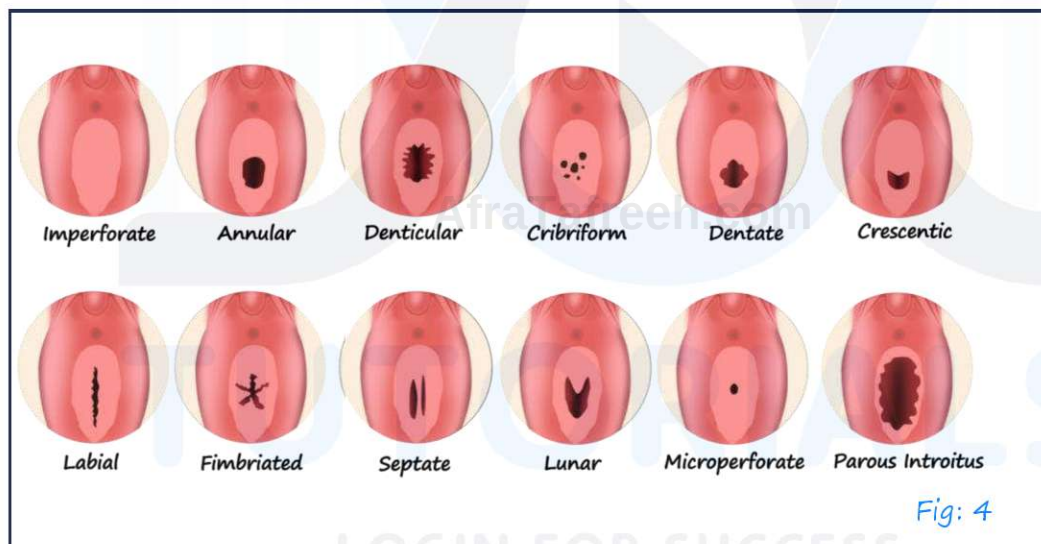
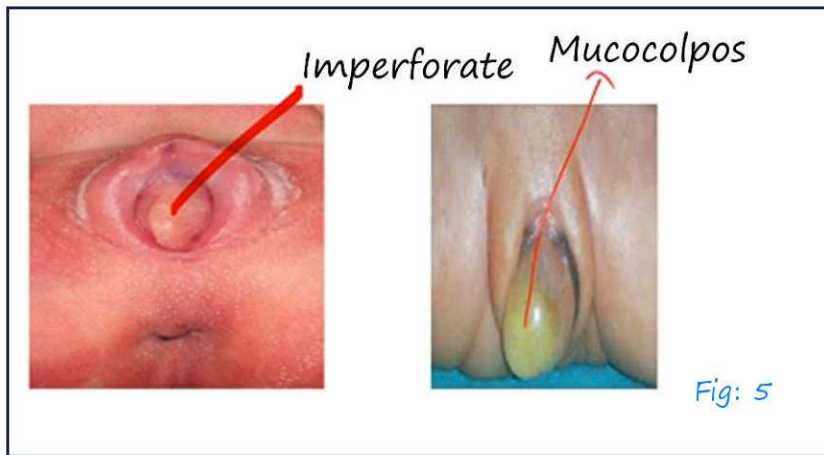
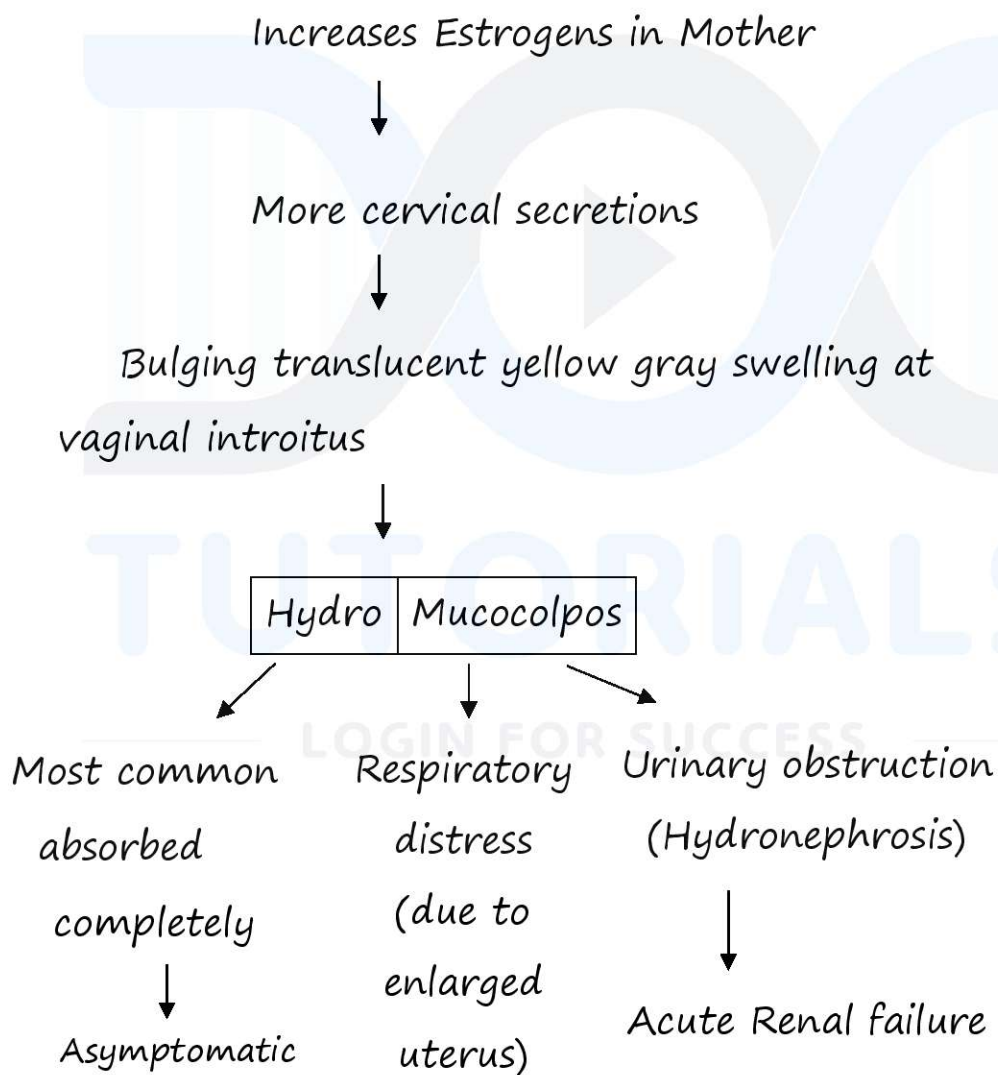


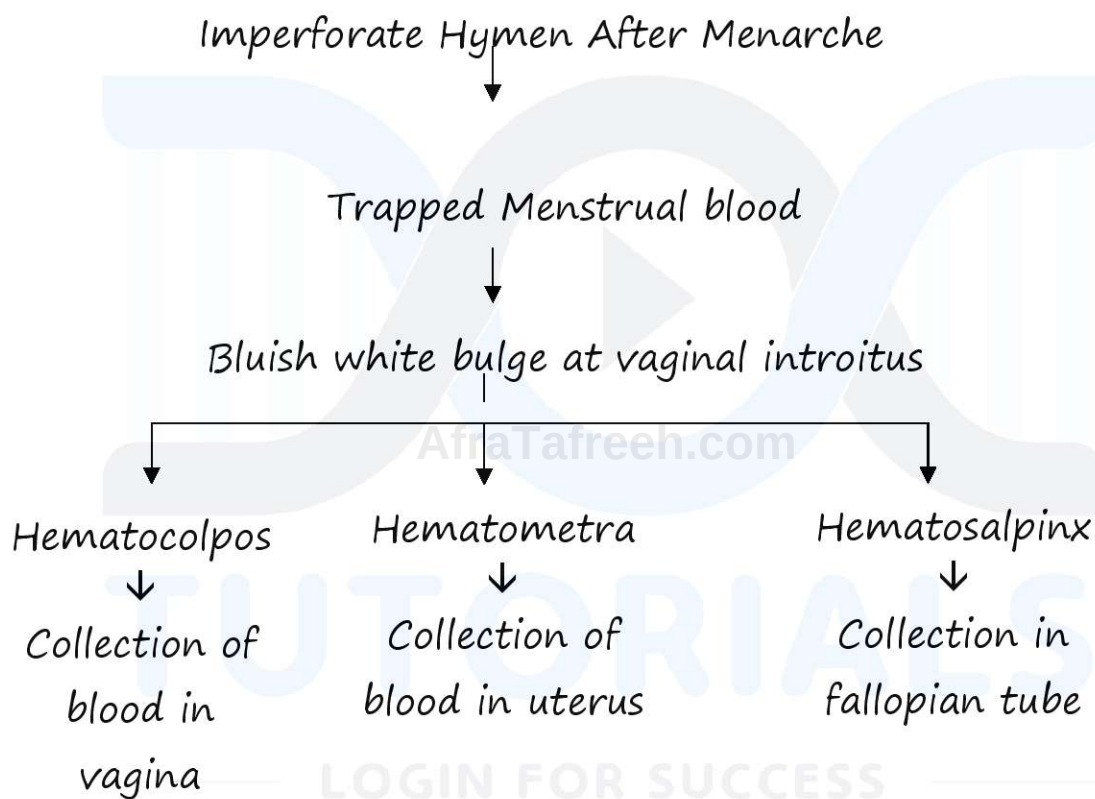
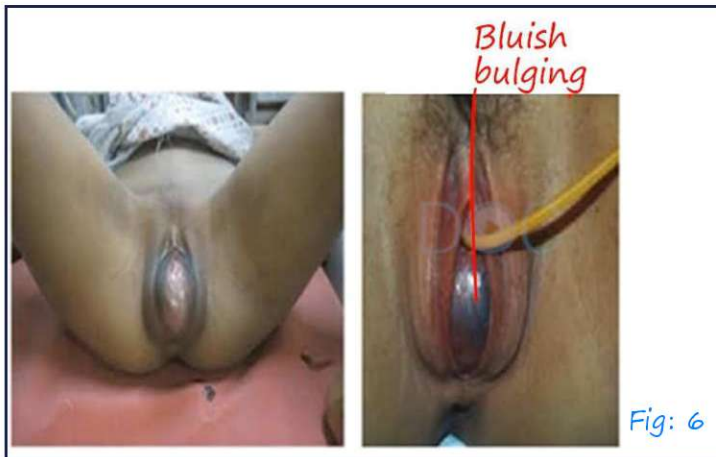
Fig: 4

- Out of these, imperforate hymen is the **most symptomatic one which needs treatment**
- After child birth, Hymen is replaced by Caruncle Myrtiliform.



i. Imperforate hymen in Newborn





Clinical Features of Imperforate Hymen. 00:15:41

- i. Primary Amenorrhea with normal secondary sexual characters
- ii. Acute abdominal pain (N) breast/ Axillary / Pubic hair
- iii. Cyclical abdominal pain
- iv. Difficulty in urination and defecation

v. Endometriosis

- Investigation of Choice – Trans abdominal ultrasound
- Gold Standard Inv – MRI

Treatment.

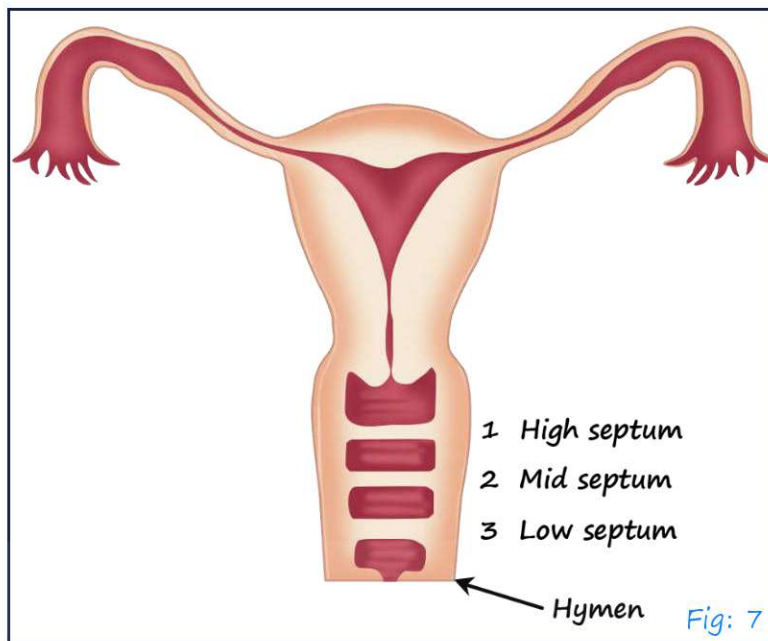
00:17:56

- i. Cruciate (star shape) incision at base of hymen
- ii. Hymenectomy – Usually done before menarche and after thelarche
- iii. These procedures are done under aseptic precautions & antibiotic cover to prevent infection.

Transverse Vaginal Septum.

00:20:01

- i. Due to failed mullerian duct fusion or failed canalization of vaginal plate
- ii. Clinically presents as
 - Primary Amenorrhea with normal sexual Characteristics
 - Hematometra, Hematosalpinx and Hematocolpos
 - On examination, thick pinkish septum seen at vaginal introitus
- iii. Incidence 1 in 70,000
- iv. Inv of Choice – trans abdominal ultrasound
 - Gold Standard – MRI
- v. Treatment – Excision of septum and forming outflow tract

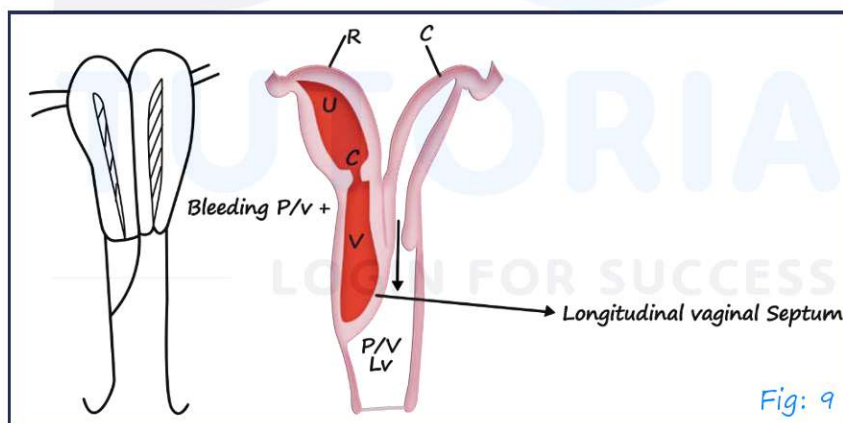


- Longitudinal vaginal septum is mostly associated with “Uterine didelphys”

Uterine Didelphys.

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00:21:58



- Complete non-fusion of Mullerian duct from proximal to distal end.
- As lower Vagina develops from sinovaginal bulb, it is normal.
- As a result of uterine didelphys, one horn of uterus & cervix is obstructed by longitudinal vaginal septum

leading to Hematometra. While the patent horn of uterus functions normally with cyclical menses.

- Right horn – Blocked
- Left horn – patent (Cyclical menses occurs)

“OHVIRA Syndrome”.

00:23:11

- Associated with “uterus Didelphys” → Longitudinal Vaginal Septum

O → Obstructed

H → Hemi

V → Vagina

I → ipsilateral

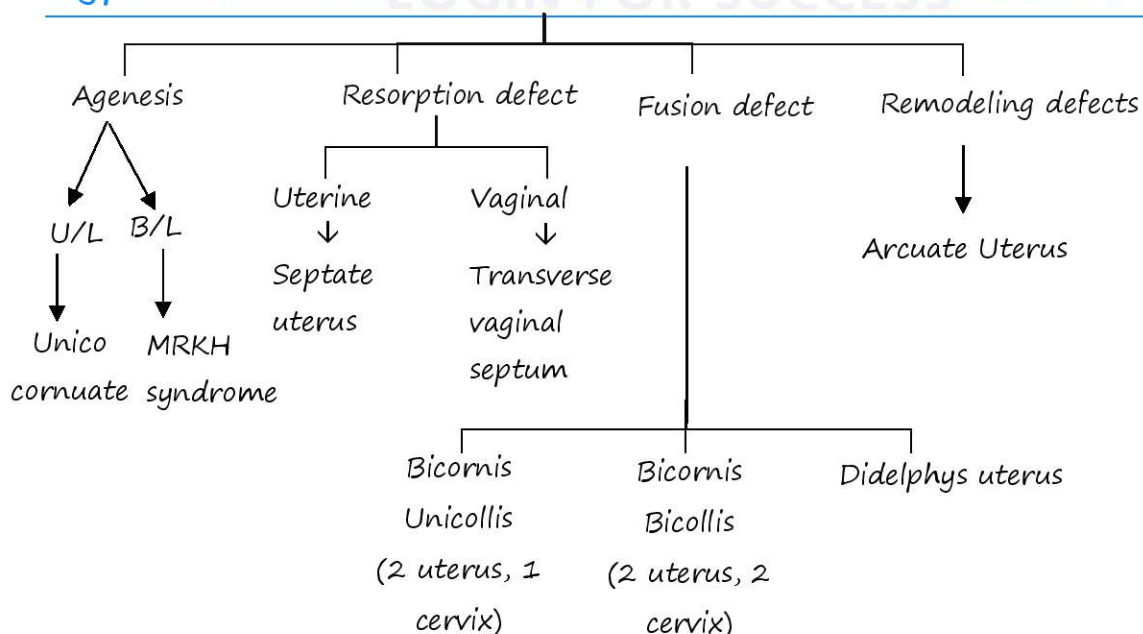
R → Renal

A → Agenesis

- Also called as “HERLYN WERNER WUNDERLICH” Syndrome

Types of uterine anomalies

00:00:24



- i. Incidence of Mullerian Anomalies – 0.5% of general population
- ii. Associated with renal anomalies – 30 – 50%
 - **Most common in U/L renal Agenesis**
 - Severe renal hypoplasia
 - Horse shoe kidney
 - Pelvic kidney
 - Ectopic / Duplicate ureter
- iii. Associated Spinal Anomalies – 10 to 12%
 - Vertebral bodies
 - Wedge
 - Supernumerary
 - Asymmetric
 - Rudimentary vertebrae
- iv. Ear Anomalies / hearing loss – 25% incidence
- v. **MURCS:**
 - MU → Mullerian Anomaly
 - R → Renal agenesis
 - CS → Cervicothoracic Somatic Dysplasia

Unilateral Agenesis.

00:30:05

- Absence or failure of development of one mullerian duct
- Condition with one fallopian tube / one uterus/ one cervix and vagina and small rudimentary horn of uterus.

Rudimentary horn	i. No Cavity in Rudimentary horn. ii. Communicating with vagina iii. Non-Communicating
------------------	--

- 40% of Renal Anomalies on affected side
- Uterus is **banana shaped** with reduced uterine cavity, cervical incompetence leading to repeated miscarriages.
- Complications → Sometimes ectopic pregnancy may occur in rudimentary horn which is non communicating
- Treatment → Salpingo oophorectomy of rudimentary side.

B/L Mullerian Agenesis.

00:33:58

- i. Known as MRKH syndrome → Mayer Rokitansky Kustner Hauser syndrome
- ii. Congenital absence of both mullerian ducts
- iii. Clinical Features
 - Normal ovaries with normal secondary sexual characteristics (breast, axillary, pubic hair)
 - Distal fallopian tube might be present
 - Uterus, cervix, upper vagina → Absent
 - Lower vagina present (sinovaginal bulb)
 - Patient presents with primary Amenorrhea associated with renal/Skeletal anomalies

- 2 to 7 % of MRKH people can have a functional uterus with cyclical bleed (small uterus)
- Difficulty in sexual intercourse as vagina is only 1-2 inches long

Treatment:

- Sexual Function – Vaginal dilators or McIndoe reed vaginoplasty (vaginal), Vechiette procedure (abdominal)
- Uterine transplantation
- Gestational surrogacy

Fusion Defects.

00:40:16

Normal: 1 uterus, 1 cervix, 1 Vagina

Bicornis Unicollis: 2 FT, 2 uterus, 1 cervix, 1 Vagina – most common

Bicornis bicollis: 2FT, 2 uterus, 2 cervix, 1 vagina

Diadelphys: 2FT, 2 uterus, 2 cervix, 2 vagina →

Associated with Longitudinal vaginal septum

Resorption / Regression defects.

00:43:39

- Vaginal Regression defects at different levels leads to transverse vaginal septum.
- Uterine Regression defects involves normal uterine cavity with septum in between called Septate uterus

Remodelling defects.

00:44:29

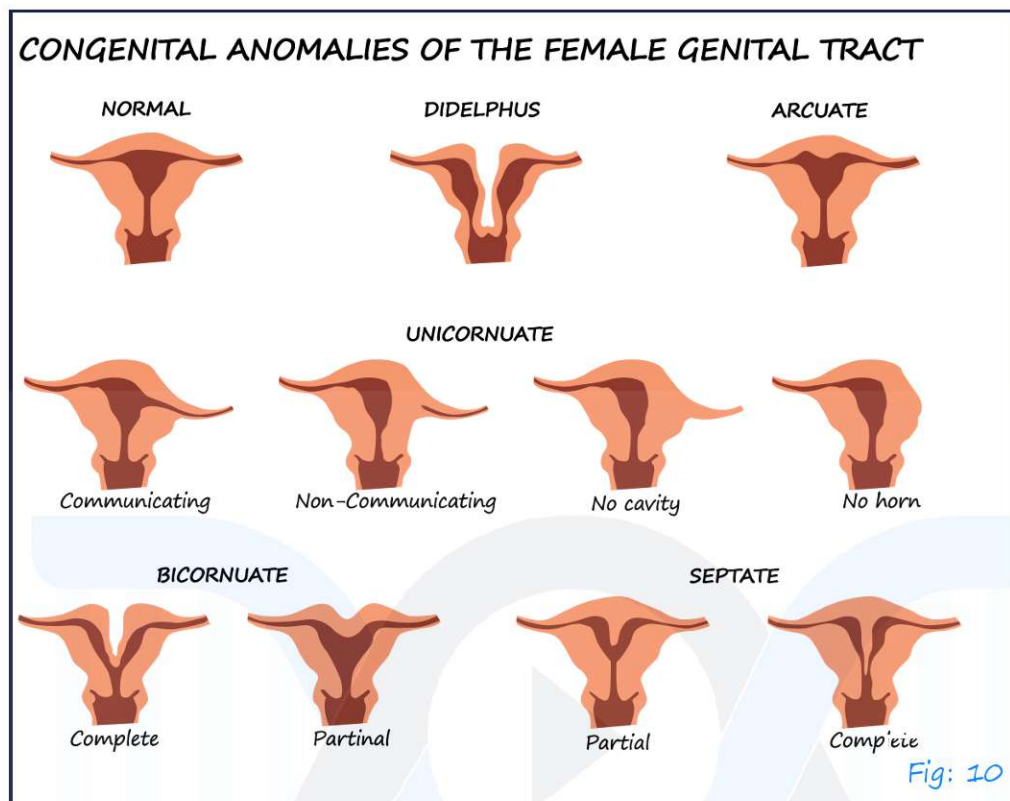
- Arcuate uterus → Most common in normal variant uterine fundus – concave with indentation < 1cm broad fundus

Diethylstilbestrol (DES) Related.

00:45:27

- DES – Synthetic estrogen which was used during pregnancy to reduce Hyperemesis.
- Female foetus which was exposed to DES in utero develops → T shaped uterus
 - Clear cell Adenocarcinoma – Vagina & cervix
 - VIN (Vaginal intraepithelial neoplasia)
 - CIN (cervical intraepithelial neoplasia)
 - Cockscomb cervix
 - Short / narrow fallopian tubes
 - Absent Fimbriae
- Male foetus exposed to DES → Cryptorchidism
 - Testicular Hypoplasia
 - Microphallus
 - Hypospadiasis
 - Reduced fertility
- Increased chances of
 - Abortions,
 - preterm labor,
 - ectopic pregnancy,
 - breast cancer,

premature menopause



Miscellaneous.

00:51:35

- i. Uterine anomalies mostly are asymptomatic / Silent
- ii. Lady can present with infertility (unable to conceive), abortion, repeated miscarriages (m/c), preterm labor, PROM (premature membrane rupture), PPRM, IUD, IUGR/malformed uterus, Placenta previa
- iii. Most common symptomatic anomaly is septate uterus (m/c) > Bicornuate uterus
- iv. Least common anomaly DES related > Didelphys.
- v. Least symptomatic Anomaly → Arcuate uterus > Diadelphys uterus
- vi. Most symptomatic → Septate uterus

vii. Inv of choice for suspected anomaly → HSG

(Hysterosalpingography)

viii. HSG cannot differentiate between septate uterus and Bicornuate uterus.

ix. Gold standard investigation is MRI

HSG and MRI findings of both septate & bicornuate uterus

	Bicornuate	Septate	
Intercornual Angle	>105°	<75°	HSG finding
Intercornual Distance	>4 cm	<4 cm	
Intrafundal downward cleft	>1 cm	<1 cm	MRI finding
Treatment	Strassman Metroplasty	Hysteroscopic septal resection	

i. Arcuate uterus does not require any treatment. It is just a variant of normal uterus.

ii. Unicornuate uterus with rudimentary horn, excision of horn is done to prevent ectopic pregnancies.

Classification of Mullerian Anomalies. 01:00:15

- ASRM – American society of reproductive medicine.

Classification

- Class 1 – Segmental Agenesis
- Class 2 – Unicornuate uterus
- Class 3 – Didelphys
- Class 4 – Bicornuate uterus
- Class 5 – Septate uterus
- Class 6 – Arcuate uterus
- Class 7 – DES Related anomaly

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TUTORIALS

LOGIN FOR SUCCESS

OVULATION INDUCTION AND HYPER STIMULATION SYNDROME

Drugs for Ovulation Induction.

00:00:13

- i. SERMs – Clomiphene citrate
- ii. Aromatase inhibitors – letrozole and anastrozole
- iii. Gonadotropins – FSH, LH
- iv. Pulsatile GnRH

For ovulation induction in PCOS the DOC is letrozole

Adjuvant Drugs.

00:01:16

- i. Glucocorticoids – Prednisolone, dexamethasone
- ii. HCG
- iii. Metformin
- iv. Cabergoline
- v. Myo inositol /D – chiro inositol

Clomiphene Citrate.

00:01:38

- Non-steroidal triphenyl ethylene derivative
- SERM (selective estrogen receptor Modulator)
- Has both estrogen agonist and antagonist properties
- Metabolised by liver and excreted in stool
- Racemic mixture of enclomiphene and zuclomiphene

Enclomiphene	zuclomiphene
- Cis form - More potent - Short $t_{1/2}$ (2.5-12hrs) - More antiestrogenic - Constitutes 62% of drug	- Trans form - Less potent - Long $t_{1/2}$ (14-33hrs) - Estrogenic - Constitutes 38% of drug

M.O.A

- Competitively binds to estrogen receptor in pituitary and hypothalamus
- ↓
- Receptor's depletion
- ↓
- Increases FSH, increases LH
- ↓
- Increases follicular size

Adverse Effects

- Impaired endometrial growth
- Decreases quality of cervical mucus
- Hot flushes
- Headache
- Breast tenderness
- Pelvic pressure
- Nausea

- Visual disturbance
- Multiple pregnancy
- Ovarian hyperstimulation syndrome (OHSS)
- Starting dose – 50mg OD
- Maximum dose – 150mg OD
- Ovulation rates – 70-80%
- Cycle fecundability – 15%
- Cumulative pregnancy rates – 70-75%
- Risk of multiple pregnancies – 7-10%
- Risk of anomalies absent
- Risk of Ca ovary/breast/endometrium absent
- Rates of miscarriage decrease with clomiphene citrate as it increases FSH and progesterone

Letrozole and Anastrozole.

00:10:09

- Triazole antifungal derivative
 - ↓
- Potent competitive non-steroidal aromatase inhibitor
 - ↓
- Blocks E_2 production
 - ↓
- Increases FSH (also increases IGF-1)
- Ovulation rates – 70-80%
- Dose – 2.5mg to 7.5mg OD

Adverse Effects

- Headache
- Cramp
- Hot flush
- Dizziness
- Fatigue
- Multiple pregnancy (3-7%)
- OHSS not seen

Gonadotropins (FSH, LH).

00:12:51

-
- Human menopausal gonadotropin (HMG)
 - Purified urinary FSH (Urofollitropin)
 - Recombinant FSH
 - Recombinant LH
 - Recombinant HCG
 - Incidence of multiple pregnancy with FSH 20-40%
 - Indications for gonadotropin treatment
 - i. Hypogonadotropic hypogonadism
 - ii. Clomiphene resistance
 - iii. Unexplained infertility
 - Gonadotropins leads to maximum increase in multiple pregnancy and OHSS

Pulsatile GnRH.

00:18:02

- T $\frac{1}{2}$ (10-40minutes)
- Low doses are given intravenously every 60-90minutes
- Uses
- Hypothalamic failure
- Hyperprolactinemia
- PCOS (clomiphene resistant)
- Multiple pregnancy (5-8%)
- OHSS (1.8%)

Dopamine Against.

00:20:44

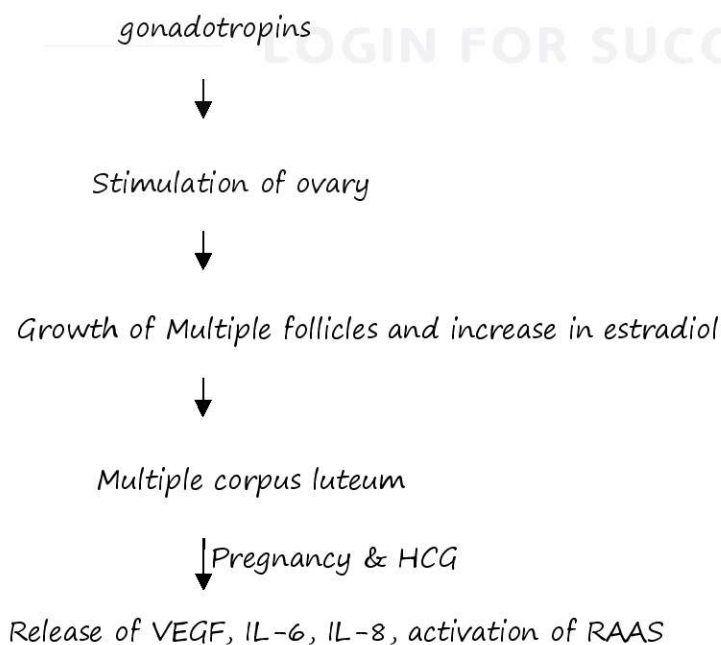
Bromocriptine	Cabergoline
Stimulates both D_1 and D_2 receptor	Stimulates only D_2
Short acting	Long acting
Orally (12hrs) vaginal (24hrs)	Works for 7 days
Less effective	More effective
More side effects	Less side effects
-	5HT _{2B} agonist
1.25 - 2.5mg daily	0.25mg thrice weekly

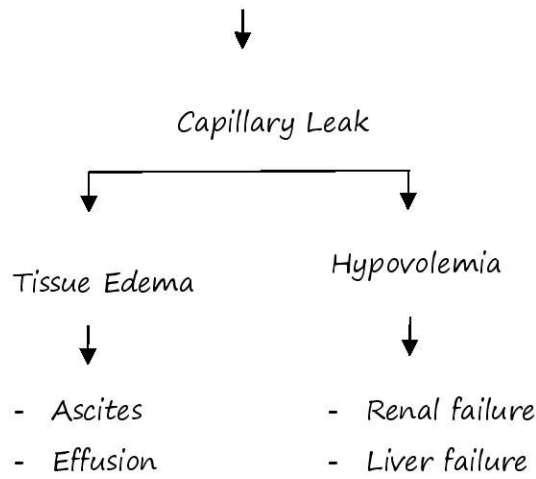
- Both are ergot alkaloids
- Both can be used for hyperprolactinemia with/without tumor
- Both can be used in galactorrhea
- Both can be used in hyperprolactinemia with PCOS
- They have less side effects when given vaginally
- They possess no risk of abortions/anomalies
- Cabergoline in high doses i.e. dose in parkinsonism leads to MR, AR, TR due 5HT_{2B} activity
- Cabergoline is the DOC in hyperprolactinemia with PCOS

OHSS.

00:28:48

- Iatrogenic complication due to FSH or clomiphene citrate
- Cystic enlargement of ovaries occurs
- Pathophysiology





Risk Factors

- Young age
- PCOS
- Increased doses of FSH
- Increased ovarian reserve
- Past history of OHSS

Predictors

- Number of follicles > 20
- Increased blood flow to ovaries in Doppler
- Increased renin
- Increased angiotensin
- Increased estradiol levels (> 2500pg/ml)

Classification

i. Mild OHSS

- Abdominal bloating
- Mild abdominal pain
- Ovarian size < 8cm²

ii. Moderate OHSS

- Moderate pain
- Nausea, vomiting
- USG evidence of ascites
- Ovarian size 8-12cm²

iii. Severe OHSS

- Clinical ascites + hydrothorax
- Oliguria (<30ml/hr or <300ml/day)
- Haematocrit (>0.45)
- Hyponatremia (Na⁺ → <135mEq)
- Hypo osmolality (< 282 mosm/kg)
- Hyperkalaemia (K⁺ >5mEq)
- Hypoproteinaemia (Albumin < 35g/L)
- Ovarian size (>12cm²)

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Critical OHSS.

00:38:19

-
- Tense ascites
 - Large hydrothorax
 - Haematocrit (> 0.45)
 - WBC (> 25,000)
 - Anuria
 - Thromboembolism
 - ARDS

Treatment.

00:38:49

- Avoid pregnancy
- Analgesics
- Antiemetic's
- Normal saline
- Calcium gluconate (to prevent arrhythmias)
- Tapping of ascites/pleural effusion
- Albumin infusion
- Anticoagulants

Prevention.

00:41:06

- Decrease dose of gonadotropins
- Decreases dose of HCG
- Cabergoline 0.5mg – has antiVEGF property
- Aspirin
- Calcium
- Metformin

MCQs

- Failure to ovulate with clomiphene citrate is called CC resistance
- CC failure is failure to achieve pregnancy even after ovulation
- Letrozole has higher pregnancy rates than clomiphene citrate.

PCOS AND ANOVULATION

Menstrual cycle.

00:00:19

Early follicular phase

- Increase in GnRH
- Increase in FSH
- Increase in Inhibin
- Increase in follicular recruitment

Mid follicular phase

- Increase in FSH stimulated aromatase activity
- Increase in Estradiol level and increase in inhibin A

Late follicular phase

- Increase in Estradiol triggers LH surge. which finally cause ovulation.

Increase E_2



increase LH (Plasminogen activator)



Ovulation

Classification of Ovulatory Disorders. 00:02:50

- **WHO group I** - pituitary or hypothalamic disorder.
 - Hypogonadotropic anovulation
 - Decrease FSH, decrease LH, decrease E_2 , Malnutrition, stress, anxiety, Weight loss, Anorexia, Exercise, Kallmann Syndrome,
 - Isolated luteinizing hormone deficiency, pituitary tumor
 - Hypothalamic tumor

- **WHO group II** – Normal gonadotropic, Normoestrogenic Anovulation?
 - In PCOS.
 - Normal FSH, E₂ & normal or high level of LH.
- **WHO group III** – hyper gonadotropic Anovulation?
 - premature ovarian failure.
 - High levels of FSH, less AMH
- **WHO Group IV** –hyperprolactinemia?
 - Prolactinoma, hypothyroidism, Drug induced hypothyroidism.
 - Decreases FSH levels, E₂ levels also decreases.
 - 75-85% Anovulation is due to PCOS (group II)
 - Group I contribute to 5-10% Anovulation
 - Group III – 10-20% Anovulation
 - Group IV- 5-10% Anovulation

Hyperprolactinemia

00:09:26

- Prolactin inhibits normal GnRH pulse
- Decreases GnRH pulse frequency.
- Decrease in FSH levels. No follicular recruitment.
- Normal prolactin level is 20-50ng/mL
- In increasing hyperprolactinemia □ will lead to short luteal phase /Anovulation /Amenorrhea. (50-100ng/ml)
- more than 100ng/mL is when complete amenorrhea occurs

Hypo & hyperthyroidism

00:11:24

- They alter the peripheral conversion of Steroids.
- Alteration in clearance of steroids
- Increase in prolactin level.

Obesity

00:13:09

- It increases Aromatase activity.
- Aromatase converts peripheral androgens to estrogens
- Increase in Aromatase $\square$ Increase in Estrogen which will inhibit FSH.
- It decreases SHBG levels $\square$ there is an increase in free testosterone, increase in free estrogen. and ultimately FSH levels drop.
- Insulin resistance and hyperinsulinemia $\square$ Increase Ovarian androgen production.

PCOS.

00:15:30

- otherwise called as stein Levinthal syndrome.

Pathophysiology

- Two important factors
 - There is a drop in the GnRH pulse frequency. because in the hypothalamus there is fall in dopamine level and there is a fall in opiod levels. which cause increase in prolactin.
 - Prolactin inhibits pituitary from releasing FSH.
- Now because of this change there is: -
 - a. Decrease in FSH
 - b. Increase in LH
- LH act on ovary and it will make the ovary secrete more androgen. Now these androgen act on liver and make the liver secrete less SHBG. (Sex hormone binding globulin). & This will lead to increase in free androgen which in turn increase LH.

- this free androgen gets converted to Estrone which acts on pituitary and inhibit the pituitary and in turn pituitary will secrete less FSH.
- Obesity leads to insulin resistance, and this leads to peripheral hyperinsulinemia which in turn stimulates the ovaries. which makes it secrete more androgen
 - As FSH is decreased $\square$ Follicular maturation is decreased so there are multiple small follicles which will secrete more AMH, more inhibin and less Estradiol.
- In turn AMH decreases FSH receptors and AMH also decreases Aromatase activity. which contributes to E_2 to decrease and increase in inhibin inhibits the pituitary.

Hormones which increase in PCOS

- Prolactin
- LH
- Androgen
- Insulin
- Estrone (E_1)
- Inhibin
- AMH
- Androstenedione
- D_1 hydro epiandrosterone (DHEA)
- DHEA – Sulfate
- 17-OHP
- CRP.

Hormones which decrease in PCOS

- FSH
- E_2
- SHBG.

Ratio between FSH/LH

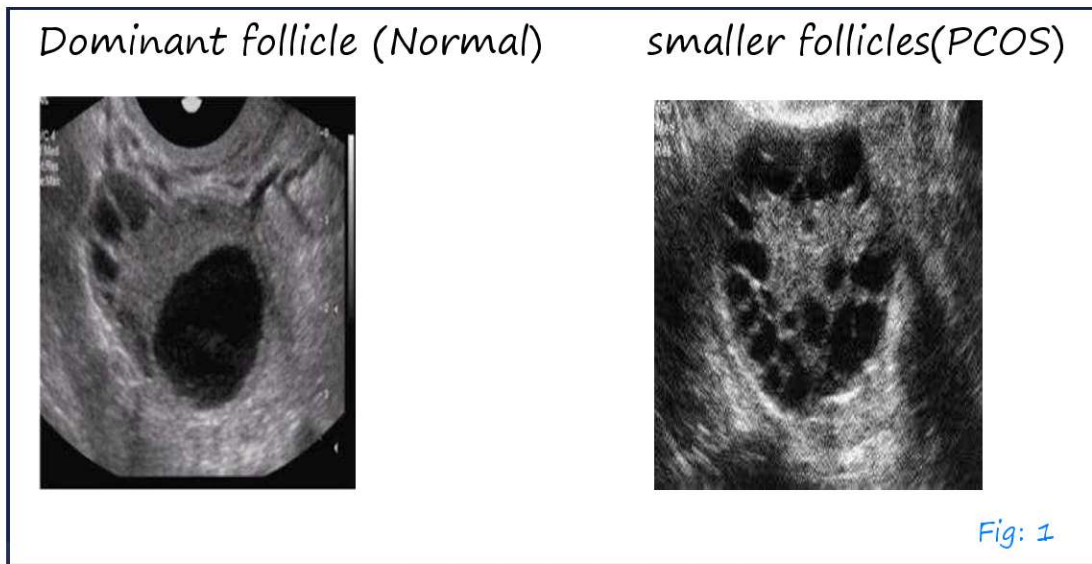
- In the beginning of the menstrual cycle, FSH should be double the amount of LH.
- LH & FSH ratio of more than 2 is marker of PCOS.

Diagnostic Criteria.

00:26:49

Rotterdam criteria

1. irregular periods
2. Hyperandrogenism □ clinical /Biochemical
3. PCO morphology in ultrasound
 - more than 12 follicles each measuring 2-9 mm. in at least one ovary.
 - Any 2 out of the three should be present to make a diagnosis of PCOS
 - In According to NIH criteria, 1&2 criteria are required.
 - According to Rotterdam criteria, any 2 criteria out of the three will diagnose PCOS.
 - According to AE-PCOS criteria. either one and 2 or 2 and 3. 2 is must.
 - Rotterdam criteria is the most commonly followed criteria.



- Follicles are arranged at periphery in PCOS
- NIH - institute of health criteria
- AEPCOS criteria - androgen excess PCOS criteria

Clinical hyperandrogenism is characterized by.

00:31:15

- i. Hirsutism -most common form.
- ii. Acne
- iii. Baldness
- iv. Clitoromegaly
- v. Voice change
- vi. Breast atrophy.

Types of androgens and their sources

1. Androstenedione - 50% from ovary, 50% - Adrenal
2. Testosterone - 25% ovary, 25% Adrenal, 50% peripheral tissue
3. DHEA - 50% Adrenal gland, 25% ovary, 25% peripheral tissue
4. DHEA - S - 100% Adrenal gland.

Androgens in PCOS and their sources

- i. Androstenedione – 60 % ovary ,40% Adrenals
- ii. Testosterone – 60% ovary, 40% peripheral conversion

Insulin.

00:35:27

- i. Potentiates Actions of LH on ovary.
 - ii. directly stimulates the ovary
 - iii. decreases SHBG production.
 - Insulin resistance in obese PCOS – 70%-80%
 - Insulin resistance in lean PCOS – 20%-25%
-
- i. Fasting insulin – $>20\text{mu/ml}$
 - ii. 1hour post meal insulin – $> 80\text{ milliu/ml}$
 - iii. 2 hours post meal insulin $\rightarrow 55\text{ milliu/ml}$
- } Insulin Resistance
- Fasting glucose /Insulin Ratio: – less than 4.5mu/ml is a marker of insulin Resistance.

Long term complications.

00:41:00

- Increase in level of Estrone stimulates endometrium, causing endometrial hyperplasia & endometrial carcinoma.
 - Dyslipidemia.
 - diabetes (type II)
 - hypertension
 - Infertility
 - Abnormal uterine bleeding
 - Obesity
 - Cardio vascular disease
 - Stroke

Syndromes associated

- i. Corpus cancer syndrome □ Endometrial Cancer
- Diabetes, hypertension and obesity.
- ii. Metabolic Syndrome – Diabetes, hypertension, obesity and Dyslipidemia.
- iii. HAIR –AN syndrome (hyper Androgenesisism, IR – Insulin Resistance, AN – Acanthosis Nigricans).



Acanthosis Nigricans.

Differential diagnosis.

00:44:45

- late onset congenital Adrenal hyperplasia □ elevated 17alpha OHP
- Androgen secreting tumor □ Rapid onset hirsutism & increase in DHEAS total testosterone > 150 mg/ml.
- Cushing's syndrome □ Increase in 24 hours urinary cortisol
- hyperprolactinemia
- Hypothyroidism
- Androgenic drugs
- Anti-seizures drug, anti-depressant drugs.

Treatment.

00:47:49

- Menstrual abnormalities □ COC /POP pills
- Hirsutism /Acne □ COC /Spironolactone/finastride /topical Eflornithine
- Alopecia □ COC/Spironolactone /finastride /Min oxidil
- Infertility –

- Letrozole	- FSH	- Metformin
- Clomiphene	Pulsatile GnRH	- Ionositol

- D chiro Ionositol: Myo Ionositol
- Laparoscopic ovarian drilling
- Vit D

Metformin.

00:51:36

- increase insulin sensitivity
- decreases weight & BMI
- decreases BP
- decreases LDL, decrease NAFLD
- decreases inflammation
- increases ovulation rates

Vitamin-D.

00:52:54

- increase insulin sensitivity
- Improves lipid profile
- Decrease Androgen levels
- decrease Inflammatory markers

Other drugs

- Statins/ Aspirin □ decrease CVS Risk – not given to women anxious to conceive
 - Rosiglitazone – increase insulin sensitivity
 - GLP – 1 analog
 - DPP – 4 inhibitors
 - COC for menstrual abnormalities only.
- Drugs under trial.
- Avoid in women
Anxious to conceive



PRIMARY AMENORRHEA

Development of External Genitalia

00:00:15

- External genitalia are Bipotential up to 9wks of IUL.
- <9 wks → Both male and female external genitalia look similar
- At 8-9 wks → secretion of testosterone begins

Testosterone

↓ ← 5α - reductase

Dihydrotestosterone

- All fetuses have inherent capacity to develop into female genitalia but upon action of hormones, they differentiate.

Male external genitalia	Female External genitalia
Scrotum	Labia majora
Glans penis	Labia minora
	Clitoris

Biopotential genitalia

↓ DHT (Androgen)

Male external genitalia.

Labia majora → scrotum

Clitoris → Penis

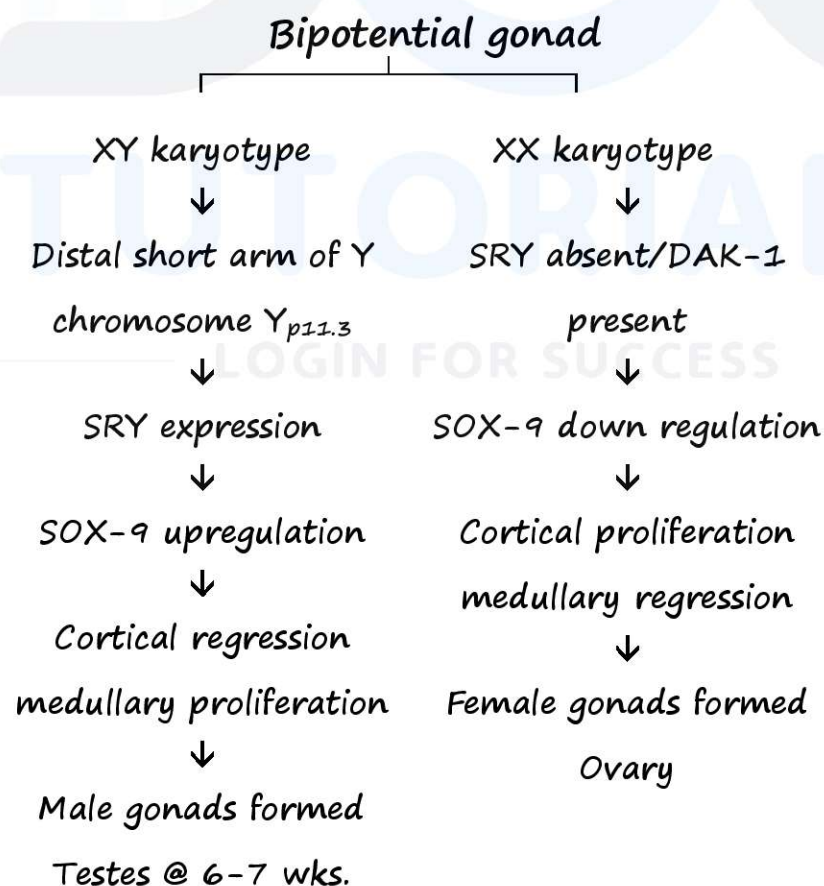
Imp MCQ's

- External genitalia is bipotential up to - 9wks IUL
- Testosterone secretion begins @ 8-9 wks IUL
- Masculisation begins by → 10 wks
- Development of external genitalia is completed by → 12-14 wks
- Crucial period to prevent masculinisation in fetuses at risk for CAH (Congenital Adrenal Hyperplasia) → 9-14 wks

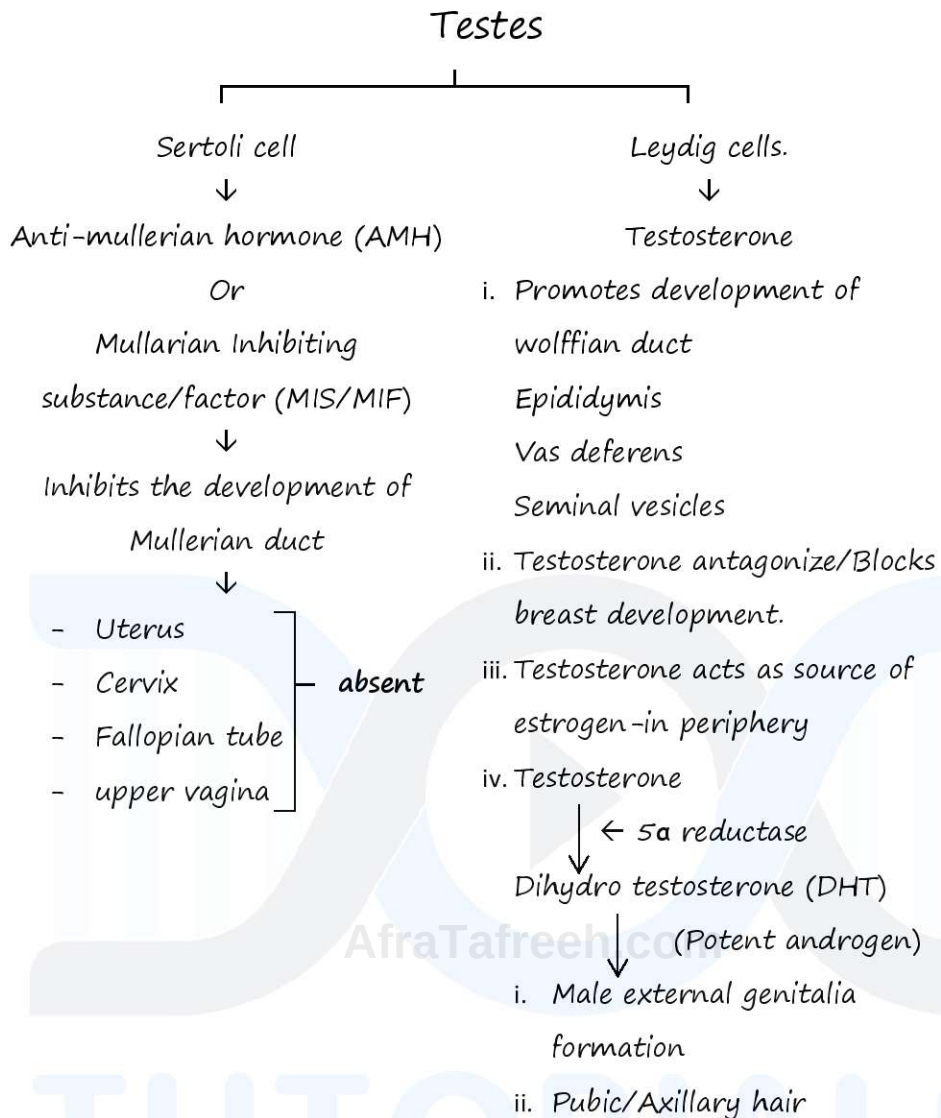
Development of Gonads

00:17:10

- Undifferentiated bipotential gonad → up to 5wks IUL.



In males



In female fetus

- Karyotype → XX
- Gonad → Ovary

Antimullarian hormone decreases ↓ Mullarian duct will grow ↓ Uterus Cervix Fallopian tube Upper vagina	Testosterone decreases ↓ i. Wolffian duct regress ↓ Seminal vesicle Epididymis Vas deferens	DHT decreases ↓ No masculinisation of External genitalia ↓ External genitalia look like females
Develop ←	Absent ← ii. Breast development seen	

Hormone important for →

i. Pubic/Axillary hair → DHT

ii. Breast → Estrogen/Progesterone → support
Testosterone → Opposes

iii. Development of external genitalia → DHT

DHT ⊕ → Male external genitalia

DHT ⊖ → Female external genitalia.

iv. Development of internal genitalia →

AMH/Testosterone

AMH ⊕ / Testosterone ⊕ → Mullerian duct ⊖

Wolffian duct grow.

AMH ⊖ / Testosterone ⊖ → Mullerian duct ⊕

Wolffian duct regress.

v. Development of breast → Estrogen/Progesterone.

E/P ⊕ / Testosterone ⊖ → Breast grow

E/P ⊖ / Testosterone ⊕ → Breast ⊖

Genes:

- Genes involved in development of testes → SRY

SOX-9

Fibroblast growth factor-9 (Fgf-9)

- Genes involved in development of ovary → Wnt-4

R – spondin

DAX-1

FOXL-2

β-catenin

- Genes involved in SRY activation:

- Steroidogenic factor (SF-1)

- Wilms tumor - 1 (WT-1)

- GATA - 4

AfraTafreeh.com

Female sex reversal

XY karyotype

+

Mutation (depletion) in genes involved in development of testes

&

Genes involved in SRY activation

↓

Female gonad

Male sex reversal

XX karyotype

+

Activation/duplication of *sox-9* (genes involved in development of testes)

↓

Male gonad

Genes involved in development of Mullerian duct:

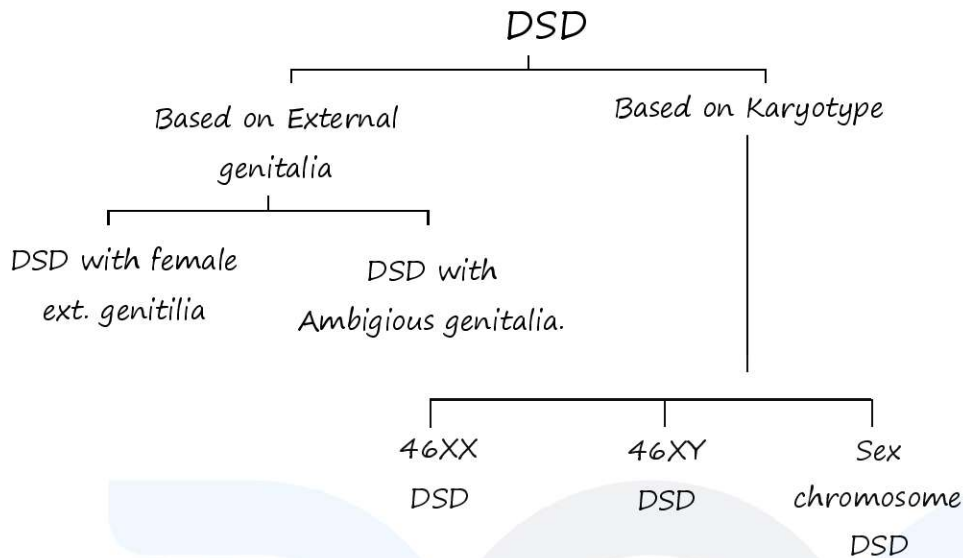
- Gene involved in
 - Selection → *Lim - 1*
 - Invagination → *Wnt-4*
 - Elongation → *Pax - 2*
 - Pax - 8*
- Any mutation of these genes → Mullerian agenesis

Genes involved in Regression of Mullerian duct:

- *Wt-1*
- *Wnt-7a*
- AMH-receptor 1 & 2
- β catenin
- *MMP - 2*

Disorders of sexual development (DSD) 00:54:13

- Includes any → Disorder of genital tract/gonad.



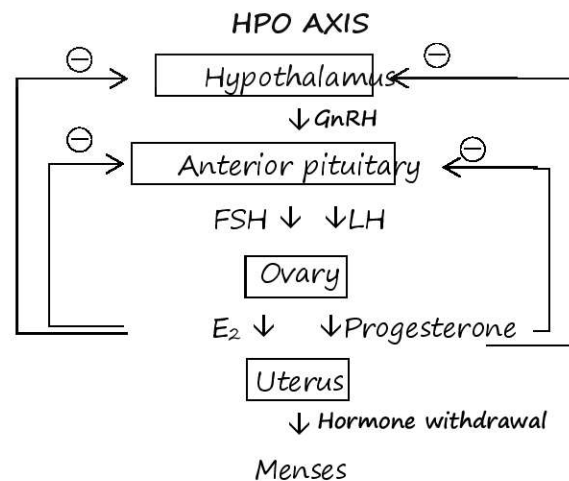
DSD with female external Genitalia:

Primary Amenorrhea

- Definition:

Absent secondary sexual characters by 13 yrs

Absent menarche by 15 yr regardless of the presence of secondary sexual characters.



Causes of Primary Amenorrhea

01:07:00

Compartment – I (Genital tract) or (End organ)			
	E/P	FSH	
i. Imperforate hymen	N	N	Eugonadotrophic Hypogonadism
ii. Transverse vaginal septum			
iii. Vaginal agenesis			
iv. Bilateral Mullerian agenesis/MRKH			

Compartment – II (Ovary)				
		E/P	FSH	
i. Turners syndrome	Non functioning Gonad	↓	↑	Hypergonatrophic Hypogonadism
ii. Pure gonadal dysgenesis				
iii. Swyer syndrome				
iv. Savage syndrome				

v. Testicular feminising syndrome (or) Androgen Insensitivity Syndrome		N	N	
--	--	---	---	--

Compartment – III (Pituitary)			
	E/P	FSH	
Craniopharyngioma	↓	↓	Hypergonatrophic Hypogonodism
Pituitary			
Compartment – IV (Hypothalamus)			
Kallman syndrome Lawrence moon Biedl Forlich			

LOGIN FOR SUCCESS

Imperforate hymen – Cryptomenorrhea.

i. Bulging bluish membrane → Hymen

ii. Accumulation of menstrual blood in

Vaginal → Hematocolpos

Cervix } → Hematometra

Uterus }

Fallopian tubes → Hematosalpinx

↓

May lead to Endometriosis

Clinical Features

- i. Abdominal pain
 - Cyclical pain (or) Persistent pain
 - Acute pain – Due to Urinary retention
Fecal retention.
- ii. Bulging bluish membrane at introitus.

Diagnosis → Clinical

- IOC → USG
- Gold standard Investigation → MRI
- Treatment → Cruciate incision
→ Star shaped/ 'X' shaped incision given.

Transverse vaginal septum

- Transverse septum in vaginal causing obstruction to menstrual flow.
- It leads to crypto menorrhea.

Clinical features:

- Similar to imperforate hymen except for bulging bluish membrane.
- IOC – USG
- Gold standard Investigation – MRI
- Treatment: Excision of septum.

Bilateral Mullerian agenesis/MRKH:

- MRKH → Mayer Rokitansky Kustner Syndrome

- B/L absence of Mullerian duct.



Uterus	}	Absent
Cervix		
Fallopian tubes		
Upper vagina		

- Present with primary amenorrhea

- It is associated with

Renal anomalies → 40%

Skeletal anomalies → 12%

- Lower vagina → present → as it develops from urogenital sinus.

Estrogen	}	Normal female levels.
Progesterone		
FSH		
LH		

Secondary sexual characters → will be present

Breast – Normal

Axillary/Pubic Hair – Normal

TURNER SYNDROME

Ovarian causes of primary amenorrhea 00:00:15

- i. Turners' syndrome
- ii. Pure gonadal dysgenesis
- iii. Testicular feminization syndrome

(or)

Androgen insensitivity syndrome

- iv. Swyers syndrome

(or)

46xy gonadal dysgenesis

Turners' syndrome

KARYOTYPES

45X

45X/46XX

45X/46XY

MOSAIC TURNERS

- Most common cause of primary amenorrhea^Q
- Described in 1938
- Most common cause of first trimester miscarriage^Q
- Most common mosaic turner → 45x /46xx (>75%)
- Most dangerous mosaic turner → 45x/46xy (risk of malignancy)

Clinical features:

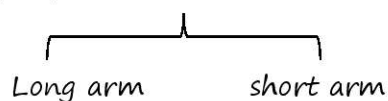
1. Short stature → due to deletion of SHOX gene
SHOX → short stature homeobox containing gene
2. Sexual infantilism → no pubertal changes
3. Low set ears
4. Webbed neck
5. Low posterior hair line
6. Widely spaced nipples
7. Short 4th metacarpals
8. Shield chest
9. Cubitus valgus
10. Lymphedema of hands and feet
11. High arched palate



Turner karyotype → 45X

Missing → X chromosome

Normally → X chromosome

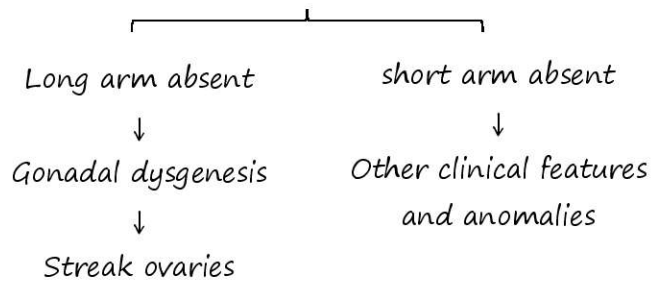


Gonadal function

In turners

Both arms of x- chromosome are missing

Missing x – chromosome



Pure gonadal dysgenesis:

Karyotype → 46xx

- Only long arm of x – chromosome is missing
- Gonadal function is absent → gonadal dysgenesis
- As short arm is present, no anomalies are seen as seen in Turners syndrome.

Turners	Pure gonadal dysgenesis
karyotype	
45x	46xx
X – chromosome	
Both long and short arm are missing	Only long arm is missing
gonadal	
Gonadal dysfunction ova – streak ovaries	Gonadal dysfunction –streak ovaries
anomalies	
Present	absent

Turners:

Estrogen	} Decreases
Progesterone	
Testosterone	

Anti mullarian hormone (AMH) – decreases

FSH, LH – Increase

Secondary sexual characters:

	Hormone responsible	IN Turners
Breast	Estrogen/progesterone	Decrease no breast development
Axillary + Pubic hair	Dihydro testosterone testosterone	Decrease no axillary and pubic hair

Uterus → Hypoplastic / infantile non-functioning uterus

(as endometrium and myometrium depends on estrogen and progesterone.

Mostly but not all turners → Primary amenorrhea.

- 15% of Turners → develop seconds sexual characters

Incomplete pubertal development

(Tanner 1, 2 stage)

- 5% of Turners → complete puberty

Begin to menstruate – followed by 2° amenorrhea.

- 30% of Turners → ovaries can be imaged.

By USG/MRI.

Anomalies and abnormalities in turners: 00:29:22

Renal anomalies:

- Most common anomaly → horse shoe kidney.

Cardiac anomalies:

- More in 45X > mosaic
- 20-30% → Aortic valve disease
bicuspid aortic valve ^Q – Most common
aortic root dilatation.
- 3-10% → coarctation of aorta ^Q

Other cardiac anomalies

1. Elongation of transverse aortic arch
2. Persistent left superior vena cava
3. Anomalous pulmonary venous return
4. Aberrant right subclavian artery (SCA)
5. Prolonged QT interval
6. Idiopathic hypertension.

Ocular abnormalities:

- Amblyopia
- Strabismus
- Ptosis
- Hypertelorism
- Epicanthus
- Far sightedness
- Red/green color blindness

Other abnormalities:

1. Endocrine abnormalities → hypothyroidism, diabetes
2. Celiac disease
3. Hearing loss
4. Liver function abnormalities
5. Attention deficit hyperactive disorder (ADHD) ^Q
6. Tumors

Increase incidence of tumors.

- CNS tumors
- Bladder cancer
- Endometrial cancer

Decrease incidence of → breast cancer.

Turners' syndrome → normal IQ ^Q

Not a cause for mental retardation

Indication of karyotyping and investigations 00:39:27

1. Unexplained short stature.
2. Delayed puberty
3. Webbed neck
4. Lymphedema
5. Coarctation of aorta.

**Gold standard investigation to diagnose turners →
karyotyping**

Investigations:

- Echo
- MRI
- Renal ultrasound
- CBC
- Thyroid function test
- Glucose – RBS
- Lipid profile
- Renal function test – (RFT)
- Liver functional test (LFT)
- Anti-endomysial antibodies
- Audiometry

1. Growth hormone

Started between 2 - and 5-years ^Q

Anabolic steroid **Oxandrolone** → between 9 and 12 years

2. Estrogen – **should not be started before 13 years**

Oral estrogen started from >13 years

↓

0.25 – 0.5mg E2 (starting dose)

↓

Increase at 3-6 monthly interval

↓

Increased Up to 2mg

↓

Decrease after 12-24 months of estrogen therapy

↓

Cyclical progesterone

5mg of medroxyprogesterone acetate (MPA)

For pregnancy → oocyte and functioning uterus needed

- For functioning uterus in turners.

Supply estrogen / progesterone therapy

↓

Uterus grow

↓

Become functioning.

For oocyte, depends on donor oocyte by IVF

In turners, pregnancy is possible by IVF. ^Q

ANDROGEN INSENSITIVITY SYNDROME

Testicular Feminization syndrome

00:00:15

- Also known as → **Androgen Insensitivity Syndrome (AIS)**
- Karyotype → 46XY
- Gonads - Testes

Dihydro-testosterone (DHT) ↓ Development of Penis, scrotum Development of Axillary and pubic hair.	Testosterone ↓ Development of Seminal vesicles Vas deferens Epididymis.
---	--

- Both Testosterone, DHT → act through same peripheral receptors chromosome responsible for these receptors

↓
Xq12

Defect in AIS:

- Inactivating mutation in androgen receptor gene (AR gene) Normally when testosterone level increases

↓ Negative feedback mechanism

LH decreases

- But here in AIS,

Due to Receptor insensitivity

↓

Pituitary is not able to sense high testosterone

↓

LH increases

↓

Testes

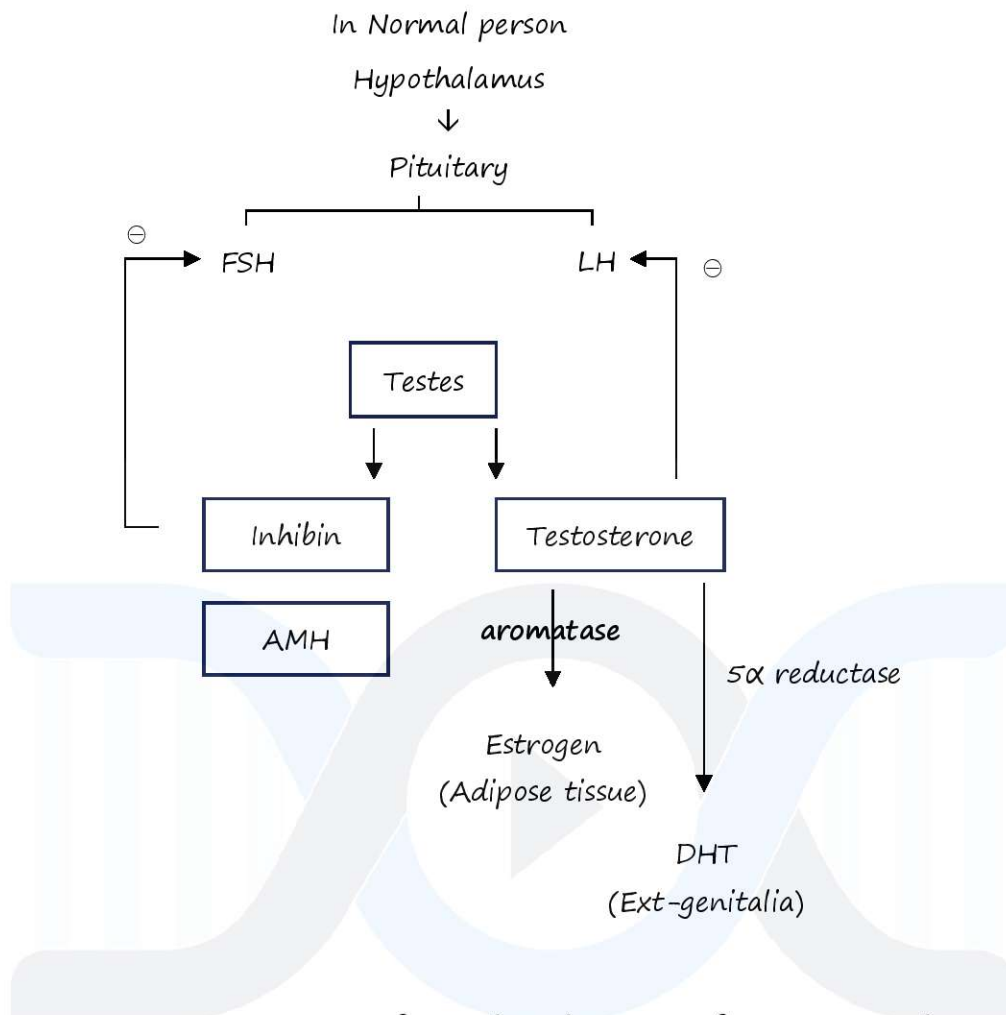
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Further increase testosterone → will not work at target organ.

Features of Testicular Feminization syndrome

00:08:35

- i. Testosterone levels increase or Normal male levels
- ii. LH → Increases
- iii. FSH → Normal
- iv. Estrogen → Increases
- v. DHT → Decreases
- vi. AMH → Normal male levels.



- Testosterone receptor function is must for conversion of testosterone to DHT in external genitalia. As testosterone receptor is insensitive DHT is not produced in adequate quantities this DHT level decreases.

Clinical features

i. Gonads → Testis

Functioning Testes



Secrete hormones

Undescended gonads Abdomen/pelvis/Inguinal canal/Labia majora -

ii. Mullerian duct → Regress ($\because$ AMH in Normal male range)

{ Uterus, FT Cervix Upper vagina	} Absent
---	----------

iii. Wolffian duct → depends on testosterone.

Here in AIS, Testosterone present but Insensitive.

So wolfian duct regresses

{ Seminal vesicle Epididymis Vas deferens	} absent
--	----------

iv. Breast → Normal development

v. External genitalia →

{ Labia Clitoris	} Female external genitalia
---	-----------------------------

- DHT is required for conversion of external genitalia from female to male. As there is inadequate ADH in androgen insensitivity syndrome conversion of external genitalia doesn't occur and external genitalia remains as female external genitalia.

Vagina → short blind pouch (Incomplete vagina)

vi. Axillary & pubic hair → absent (DHT decreases)

vii. Height → Greater than Normal woman

($\because$ Karyotype is 46xy with high estrogen levels)

Management/Treatment

00:31:00

- External genitalia → Female type
- Breast → Normal
- Vagina → short blind pouch
 - Vaginoplasty/progressive vaginal dilatation.



Mc Indoe Reed vaginoplasty

Williams vaginoplasty

- Vaginoplasty → Should be done prior to marriage.
- Pregnancy → Not possible

Gonadectomy

- Undescended gonads → Gonadoblastoma
- To be done after puberty ^Q → 16-18 year

After gonadectomy



Hormone replacement therapy can be started.

LOGIN FOR SUCCESS

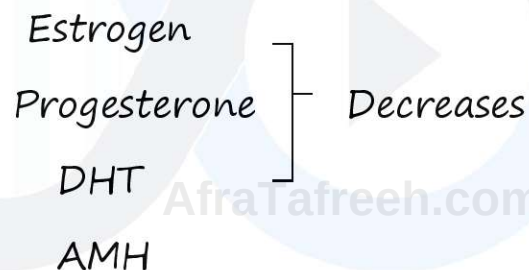
SWYERS SYNDROME

- 46XY Complete Gonadal Dysgenesis



Clinical Features

1. Hormone deficiency due to absence of functional Gonad



2. FHS/LH → Increase
3. Gonad - Dysgenetic / Streak Testis
4. Breast → Absent
5. Axillary/Pubic hair → present/Sparse (from adrenal androgens)
6. Internal genitalia
 - Wolffian Duct → Regress
 - Mullerian Duct → Present (uterus, FT, Cervix, Upper Vagina)
7. External Genitalia → Female Type

Treatment

1. Hormone Replacement Therapy

Estrogen – (start @ 13 years)

↓ 1-2 yrs

Progesterone

Estrogen, progesterone → Breast Development

Menstrual Cycle

2. Pregnancy possible by Donor Oocyte & IVF^Q

3. Gonadectomy done to avoid Gonadoblastoma as soon as possible

Swyers:

46XY with Dysgenetic Gonad with Mutant SRY Gene

↓

High Risk of Gonadoblastoma^{QQ}

↓

Risk → 20-30% ^{QQ}

In Primary amenorrhea, condition with Highest Risk & Gonadoblastoma is Swyers Syndrome

DIFFERENTIAL DIAGNOSIS

	Mullerian agenesis	Tumors/PGD	TFS X- Linked recessive	Sawyers (mutation in SRY gene)
Karyotype	46XX	45X	46 XY	46 XY
hormones	normal			
estrogen(E)	female	decrease	Increase	Decrease
Progesterone(P)	range	decrease		Decrease
Testosterone		decrease	Increase	Decrease
DHT		decrease	Decrease	Decrease
AMH		decrease	Increase	Decrease
FSH		increase	Normal	Increase
LH		increase	Increase	Increase
Breast	Present	Absent	Present	Absent
Axillary/public hair	Present	Absent	Absent	Absent
Gonads	Normal Ovaries	Streak Ovaries	Undescended testis	Streak Ovaries
Gender identify	Girl	Girl	Girl	Girl
Internal genitalia	Absent	Present	Absent	Present
uterus		Hypoplastic		Hypoplastic
vagina		uterus		
wolffian duct				
External genitalia	female	female	Female	Female

SECONDARY AMENORRHEA

Definition:

- It is absence of menstruation for 6 months or more in a woman in whom normal menstruation has been established

(or)

Absence of menstruation for 3 cycles in lady who has been regularly menstruating

Types of secondary Amenorrhea

00:09:30

-
- i. Physiological (m/c)
 - ii. Pathological

1. Physiological Secondary Amenorrhea

- Pregnancy (m/c)
- Lactation
- Menopause – secondary amenorrhea after 40 yrs age

2. Pathological Secondary Amenorrhea – secondary amenorrhea before 40 yrs age

- Polycystic ovarian syndrome (m/c)

Causes of Secondary Amenorrhea

00:16:20

1. Uterine factors (compartment -I)

- Tuberculosis (Endometritis)

- Post radiation
- Synechiae (Adhesion) → Asher man's syndrome
- Surgical removal → Hysterectomy/Endometrial ablation

2.Ovarian factors (Comportment -II)

- PCOS (m/c)
- Premature ovarian failure
- Resistant ovary syndrome (savage syndrome)
- Hyper estrogenic state
 - Metriopatheia hemorrhagic
 - Feminizing tumor of ovary (Granulosa cell tumor)

3.Androgen Excess

- Masculinizing tumor of ovary – (Sertoli Leydig cell tumor/theca cell tumor)
- Adrenal tumor /hyperplasia/Cushing's syndrome

4.Hypoestrogenic state

- Oophorectomy
- Pelvic radiation

5.Pituitary factors – (Comportment – III)

- Pituitary adenoma
- Cushing's syndrome
- Acromegaly
- Sheehan's syndrome
- Simmonds's disease

6.Hypothalamic factors (comportment – IV)

- Psychogenic shock, stern

- Anorexia nervosa
- Strenuous exercise
- Pseudocyesis
- Congenital malformation
- Trauma
- Surgery
- Radiotherapy
- Infection (Tb, sarcoidosis)
- Tumors (craniopharyngioma, meningioma)

7. Thyroid factors

- Hypothyroidism

8. General diseases

- Malnutrition, Tuberculosis
- Diabetes
- Chronic renal failure

9. Iatrogenic

- COC
- Phenothiazines
- Anti-hypertensives
- Dopamine antagonist

Mechanism of Secondary Amenorrhea 00:35:24

- Uterine factors E, P, FSH, LH - N
Mechanism → destruction of endometrium
- POCS

- Increase LH/increase Androgen

- ↓
- Increase Estrogen E_1 , increase Inhibin
- ↓
- Decrease FSH → Anovulation (No progesterone)

Progesterone challenge test

- Expose patient with progesterone



Withdraw



Bleeding occurs



Progesterone challenge test +ve

- Bleeding occurs only when hormones fluctuate
- N cycle → E_2 - Endometrium thick



Ovulation



Progesterone - (Endogenous Estrogen blocker)



Down regulate estrogen receptors

PCOS

Ovulation → does not occur

Estrogen → Estrone E_1 continuously present

- No progesterone to oppose estrogenic effects in endometrium



No fluctuation in estrogenic effects

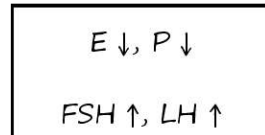
- Premature ovarian failure (PoF)

/oophorectomy/Radiation:

Absence of follicles



Anovulation /decrease Estrogenic



- Savage syndrome

Resistant ovary syndrome

Defect in FSH receptor



Resistance to gonadotropin

Follicles – do not grow

↓ Estrogen, ↓progesterone

↑ FSH

- metriopatheia Hemorrhagic

Granulosa cell tumor

Persistently high (or) same estrogen level

No fluctuation in estrogen



Endometrium keep growing



Endometrium outgrows its blood supply



Hypoxia



Sheds

- Androgen excess

Sertoli -Leydig
 Adrenal hyperplasia
 Cushing's

} androgen excess

↓

Oppose the effect of estrogen on
endometrium

- Oophorectomy

&
 Pelvic radiation

} Decrease Estrogen

- Prolactinoma

Excess prolactin → Inhibits pulsatile GnRH release –
most imp effect

Inhibits pituitary gonadotropin release
Inhibits ovarian steroidogenesis



Hypoestrogenic state

- Hypothyroidism

Decrease T3, T4



Increase thyrotrophin Releasing Hormone



Hyperprolactinemia



Hypoestrogenic state

- Malnutrition → decrease GnRH.

- Combined oral contraceptive (E+P) → decrease
GnRH

Decrease FSH, decrease LH



Anovulation

- No fluctuation in E_2 levels

- Drugs :Antipsychotic drugs → Decrease Dopamine



Hyperprolactinemia



Hypoestrogenic state

Hormonal changes

01:07:35

INICET Q

	E	P	FHS	LH	PRL
uterine factor	N	N	N	N	N
PCOS	$E_1 \uparrow$ $E_2 \downarrow$	↓	↓	↑	↑ or N
POF/oophorectomy	↓	↓	↑	↑	N
pituitary	↓	↓	↓	↓	↓
hypothalamic	↓	↓	↓	↓	N or ↑
pregnancy	↑	↑	↓	↓	↑

- Main condition with increase Estrone, decrease progesterone → PCOS

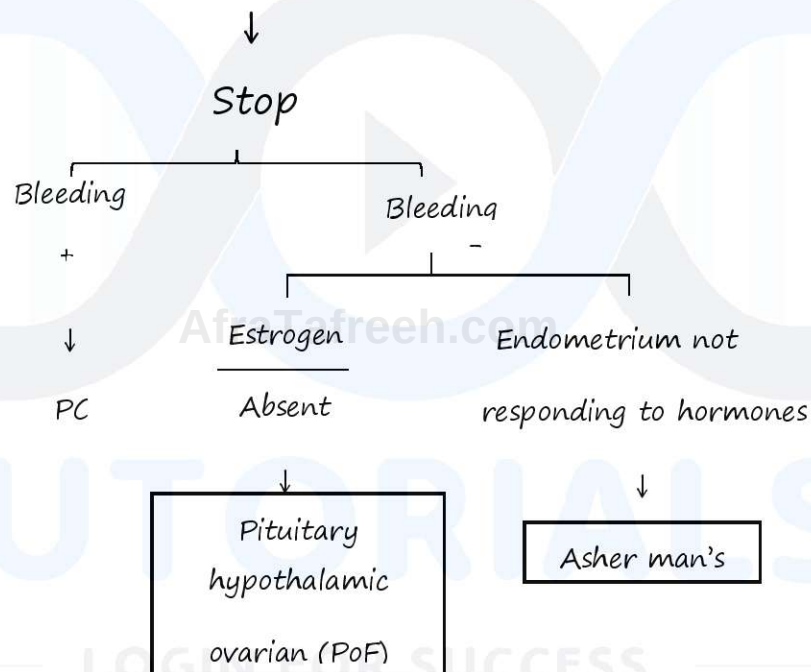
- Other conditions with increase estrogen, decrease progesterone → metropathia Estrogen secreting tumor
- In pituitary cause GnRH increases
- In hypothalamic cause GnRH decreases

Progesterone challenge test

01:19:30

- Expose lady to progesterone 7-10 days

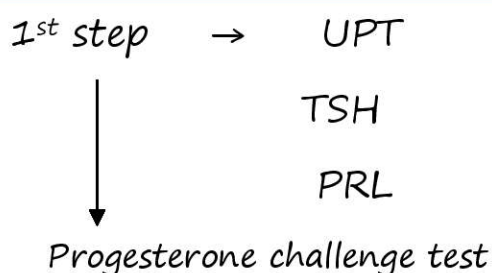
(Progesterone works only in estrogen primed endometrium)

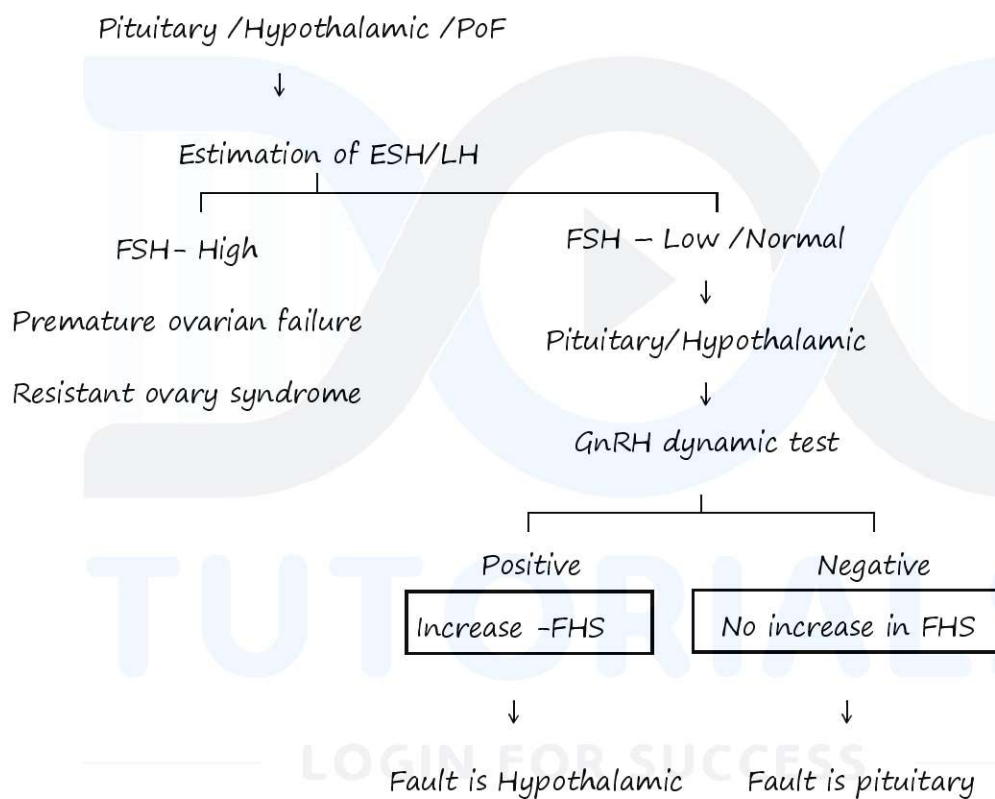
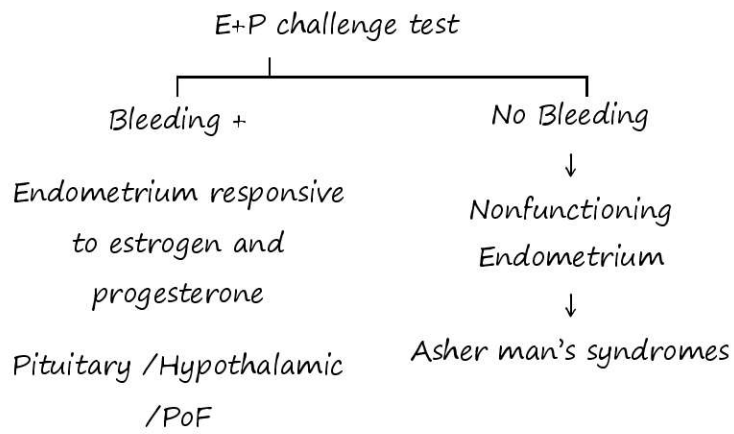


E/P → decrease

Evaluation of secondary amenorrhea

01:25:40

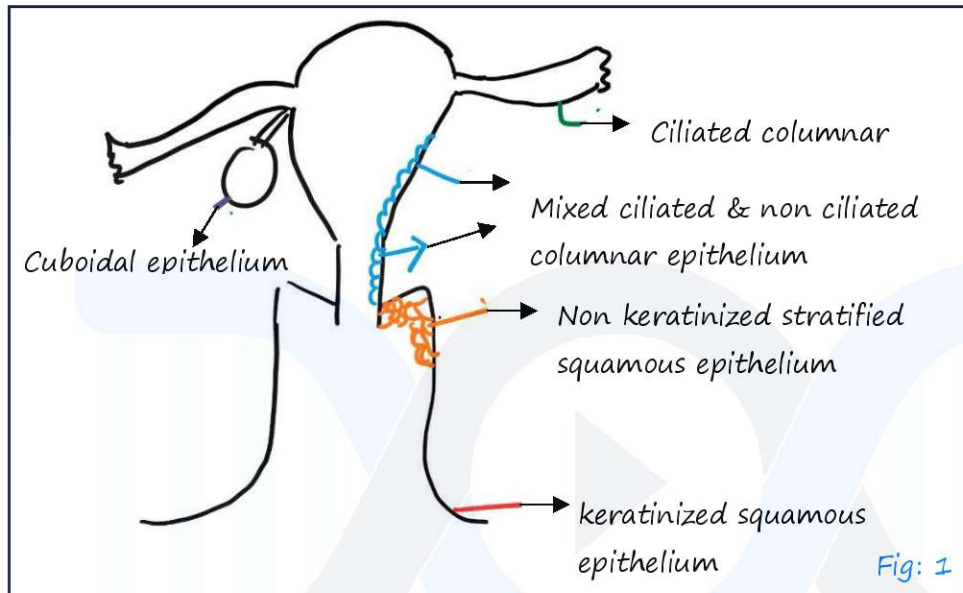




PRE-MALIGNANT LESION OF CERVIX

Types of Epithelia

00:00:47

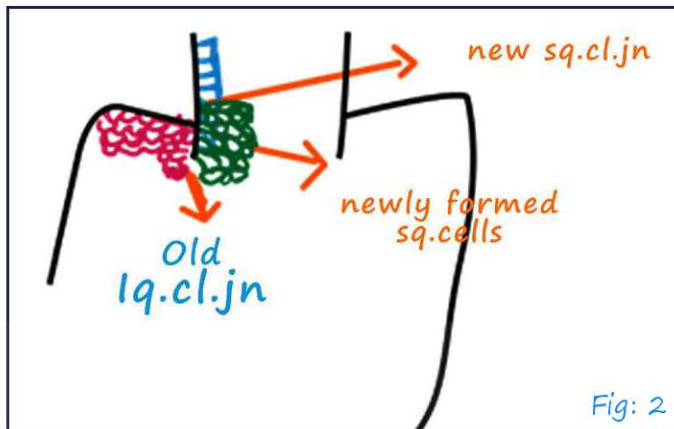


- Vulva – Keratinised stratified squamous epithelium
- Vagina & ectocervix – Non keratinized stratified squamous epithelium
- Endocervix – Mixed ciliated and non-ciliated columnar epithelium
- Endometrium – Mixed ciliated and non-ciliated columnar epithelium
- Fallopian tube – Ciliated columnar epithelium
- Ovary – Cuboidal epithelium

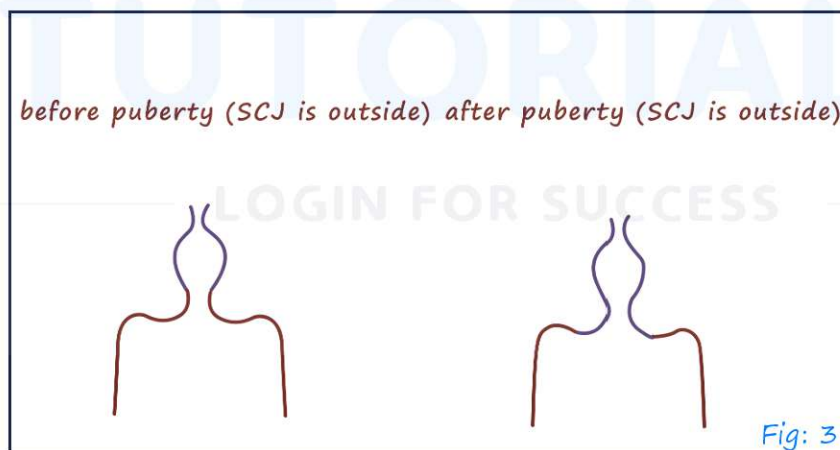
Transformation zone

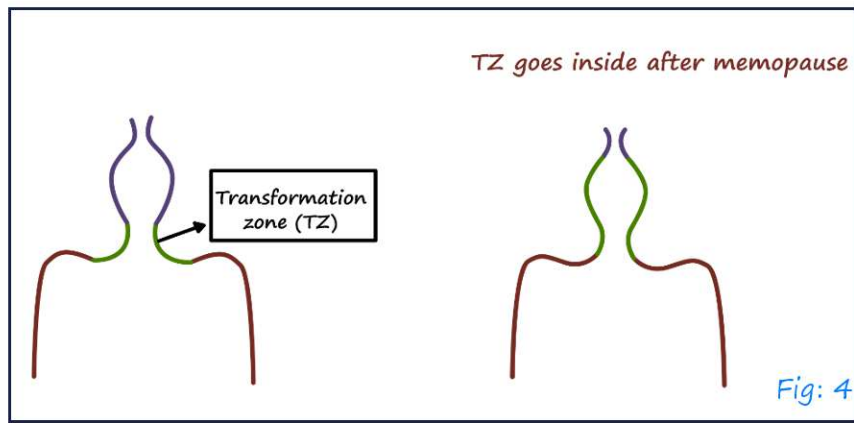
- Newly formed after puberty

- The area between old and new squamous columnar junction



- Metaplasia: When normal epithelium → Converted another normal epithelium
- Dysplasia: When normal epithelium → Converted to abnormal epithelium
- When HPV – infects → Dysplasia seen





Conditions altering squamous metaplasia

00:11:56

- Metaplasia is mainly due to hormone estrogen

Increasing conditions	Decreasing conditions
i. Adolescence	i. Menopause
ii. Pregnancy	ii. Prolonged Lactation
iii. COC ↓ Increases risk of C_x cancer	iii. POP ↓ Decreases risk of C_x Ca

Human Papilloma virus

00:14:34

- Number of subtypes → 150 subtypes
- Number of subtypes infect lower genital tract → 40
- Number of subtypes oncogenic → 15
- MC sexually transmitted disease worldwide → HPV

- MC symptomatic genital tract infection worldwide → Chlamydia
- Highest prevalence age group – 20-24yrs
- Clearance rate – 90%
- Continuation rate – 10%
- Usual time taken for clearance – 9 to 15 months
- 12 months
- MC subtype – HPV 16
- 2nd MC – HPV 18
- 3rd MC – HPV 45

Vaccination

00:19:30

	Name	Strains included
HPV 2	Cervarix/Bivalent	16,18
HPV 4	Gardasil – 4/Quadrivalent	6,11,16,18
HPV 9	– Gardasil – 9/Nanovalent	6, 11, 16, 18, 31, 33, 45, 52, 58

- Ideal age – 11-12yrs earliest – 9yrs
late – 45yrs

Doses	
< 15 years	> 15 years
2 doses	3 doses → 0,1, 6, or 0,2,6

- Vaccine efficiency – 100% effective
- Duration of protection – 6.4 years
- Sexually active women – **Vaccinate**
- During pregnancy – No
- During lactation – yes
- Immunocompromised – yes
- Screening after vaccination Continued same guidelines

Vaccine prevents –

1. Cervical cancer
2. Vaginal cancer
3. Vulval cancer
4. Oropharyngeal cancer
5. Larynx cancer
6. Penis cancer
7. Anus cancer

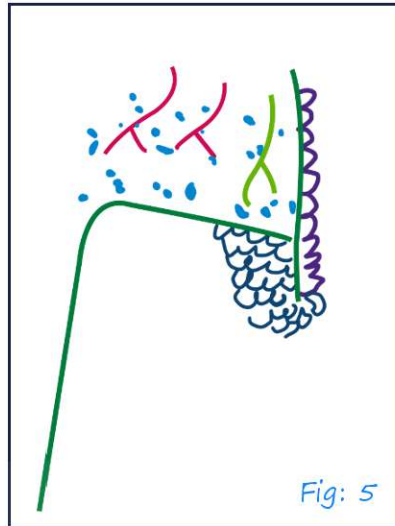
- Can HPV – vaccine be given for males? (YES)

Gardasil – 9

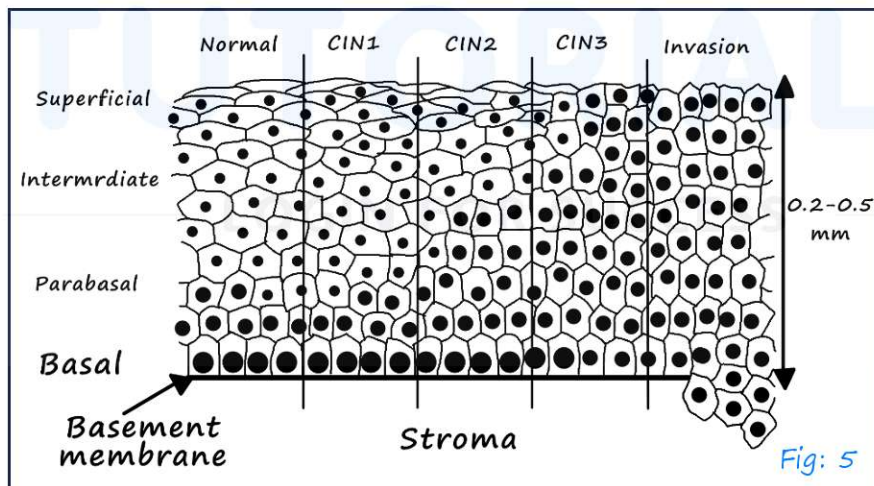
Types of CIN

00:30:24

- Cervical intraepithelial Neoplasia (Basement membrane – not invaded)



- CIN 1 – Involves the deepest layer of epithelium (lower 1/3)
- CIN 2 – Involves the middle layers (middle 1/3)
- CIN 3 – Involves the superficial layer (upper 1/3)
- Carcinoma insitu → Basement membrane not involved



Types of CIN	Malignant or premalignant	Incidence of malignancy	Regression
CIN 1	Not a premalignant	1% incidence of malignancy	57%
CIN 2	Premalignant	5% incidence of malignancy	43%
CIN 3	Premalignant	20% incidence of malignancy Or >12% incidence of malignancy	32%

Different screening techniques

- PAP
- Co-testing → PAP + HPV DNA
- HPV triage → Liquid based PAP + HPV DNA + Colposcopy
- i. PAP → 1. Conventional PAP 2. Thin/Liquid based

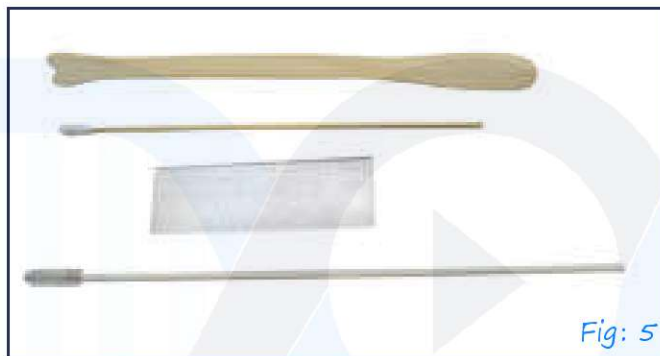
LOGIN FOR SUCCESS

Pre requisites

- Avoid sexual intercourse – 2days prior taking smear
- Avoid vaginal douching
- Avoid lubricants/P/V examination

Pap kit

- Ayer's spatula
- Endo brush / cytobrush
- Slide
- Swab
- Fixative use for PAP smear is 95% ethanol immediately before the slide dries



Sensitivity of PAP:

- Sensitivity of conventional pap smear → 50%
- Sensitivity of liquid-based pap smear → 80%
- Sensitivity of co-testing → 100%

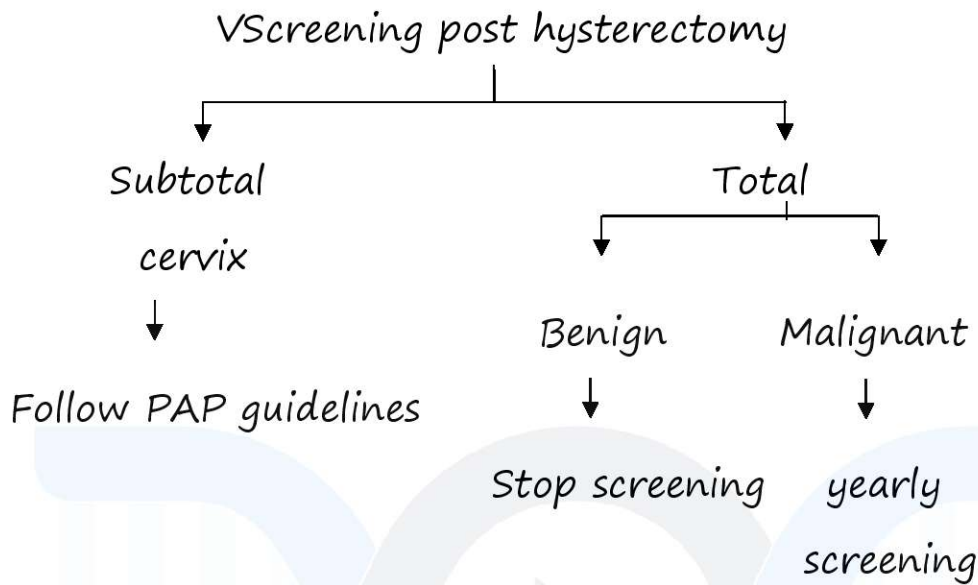
LOGIN FOR SUCCESS

Screening Guidelines

00:47:40

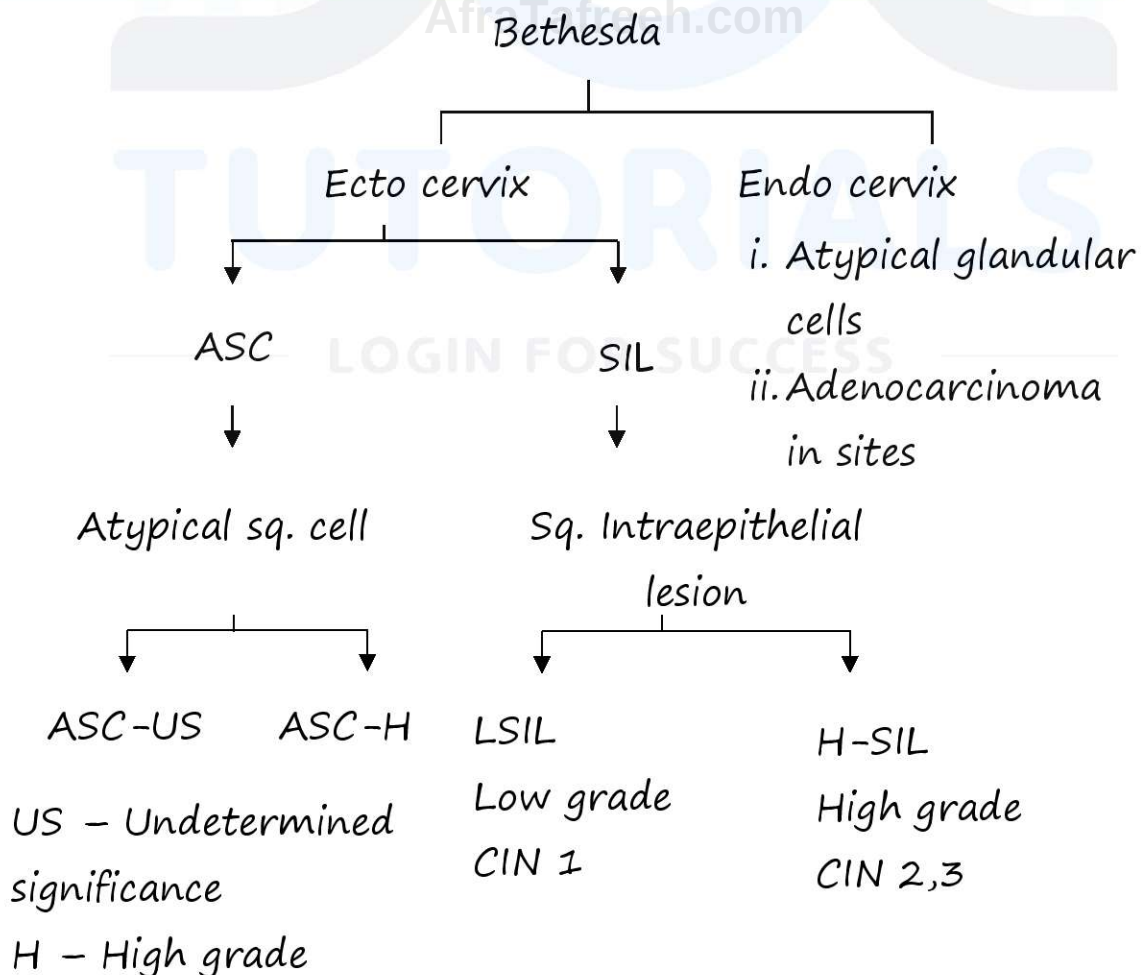
- Starting at age – 21yr
- Between 21 to 29 do cytology every 3yr
- Between 30-65 do cytology every 3yrs or contesting every 5yr
- Stop at 65yrs
 - 3 negative cytologists
 - 2 negative contesting
 } in last 10yr

- Screening after vaccination - Yes
- Screening for HIV positive → Screen, yearly



Bethesda

00:53:19



Additional diagnosis with Pap

- i. Trichomonal
- ii. Candida
- iii. Actinomyces
- iv. Bacterial vaginosis
- v. Herpes
- vi. Inflammation IUCD radiation
- vii. Atrophy

Management

01:01:01

- Women with age > 25yrs
 - ASCUS - Reflex HPV {
 - +ve → colposcopy
 - DNA
 - ve → cotest/cytology (PAP)
 - ASC-H
 - LSIL
 - HSIL
 - AGC
 - AIS
- colposcopy (Technique to see ecto cervix)
- conization

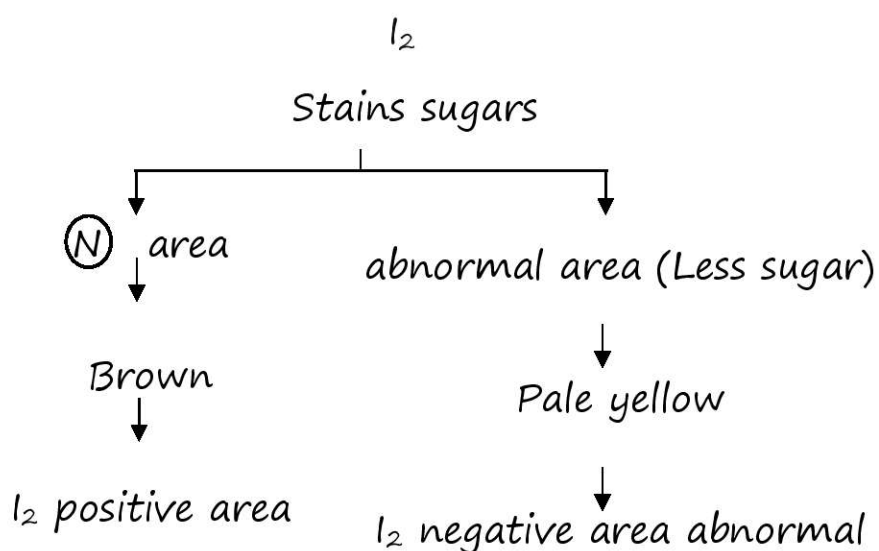
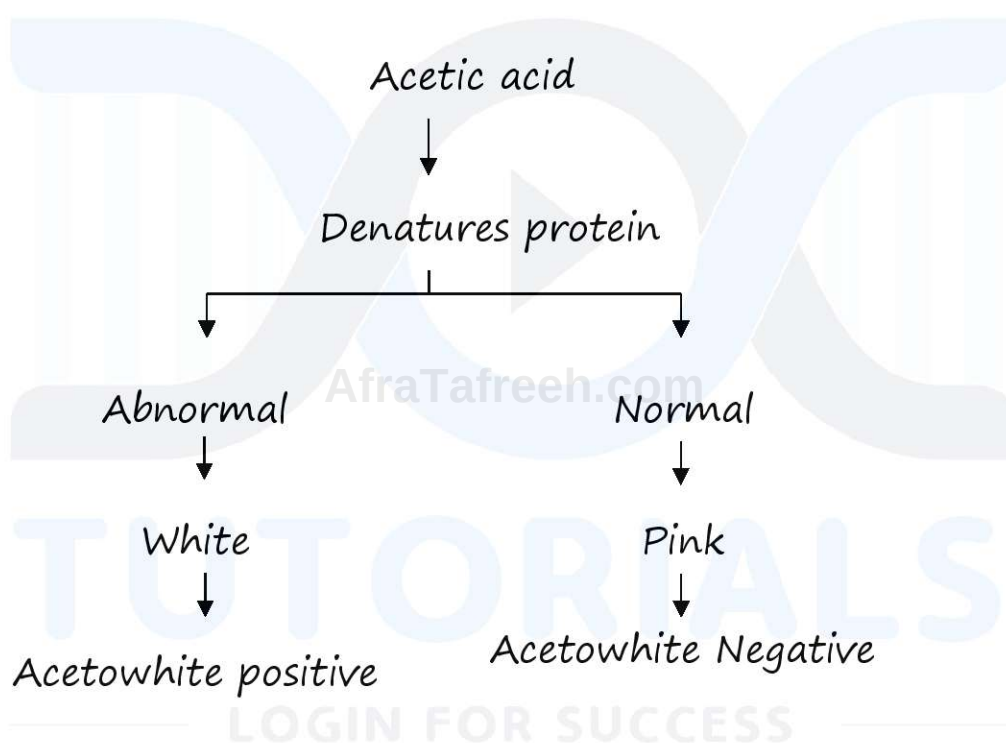
Colposcopy:

- Instrument to magnify the cervix
- Transformation zone is visualized → Ecto cervix
- Endo cervix - not visualized

- Q. Conventional colposcopy → 15-40 times magnification
- Q. Colpo microscopy → 200 times magnification

Dyes used

- Before performing colposcope → 2 dyes are used
- VIA – Visual Inspection with acetic acid
- VILI – Visual inspection with lugol's I_2



- Abnormal area
- Aceto white positive
- Iodine negative
- Visualized under colposcopy
- Biopsy done

Cryotherapy

01:12:04

- Crystallizing intracellular water using a refrigerant gas used – Nitrous oxide (MC), CO₂
- Advantage
 - Easy/OPD procedure
 - Cheap
 - Safe
- Disadvantages
 - No tissue available for HPE examination
 - Can't be used for CIN3
 - Cause Uterine Cramping
 - Vasovagal shock
 - Squamo – columnar junction – moves up
 - Used for CIN-1

CO₂ laser ablation

- Tissue destruction 5-7mm.
- Can be used for irregular CIN

- Loop Electrosurgical excision procedure
- Large loop excision of transformation zone
- Advantages
 - Safe/Easy
 - Cheap/UPD
 - Local anesthesia
 - Specimen for HPE
- Disadvantage
 - Thermal damage to the margin
 - post procedure bleeding
 - vapour fume inhalation
 - Rx OC for CIN – III → LEEP

Cold knife conisation

- Cutting cervix in cone shape. → Conisation
- Advantages
 - Therapeutic procedure
- Disadvantage
 - General anesthesia needed
 - If excised large mass → Sphincter capacity of cervix is lost → Loss of pregnancy

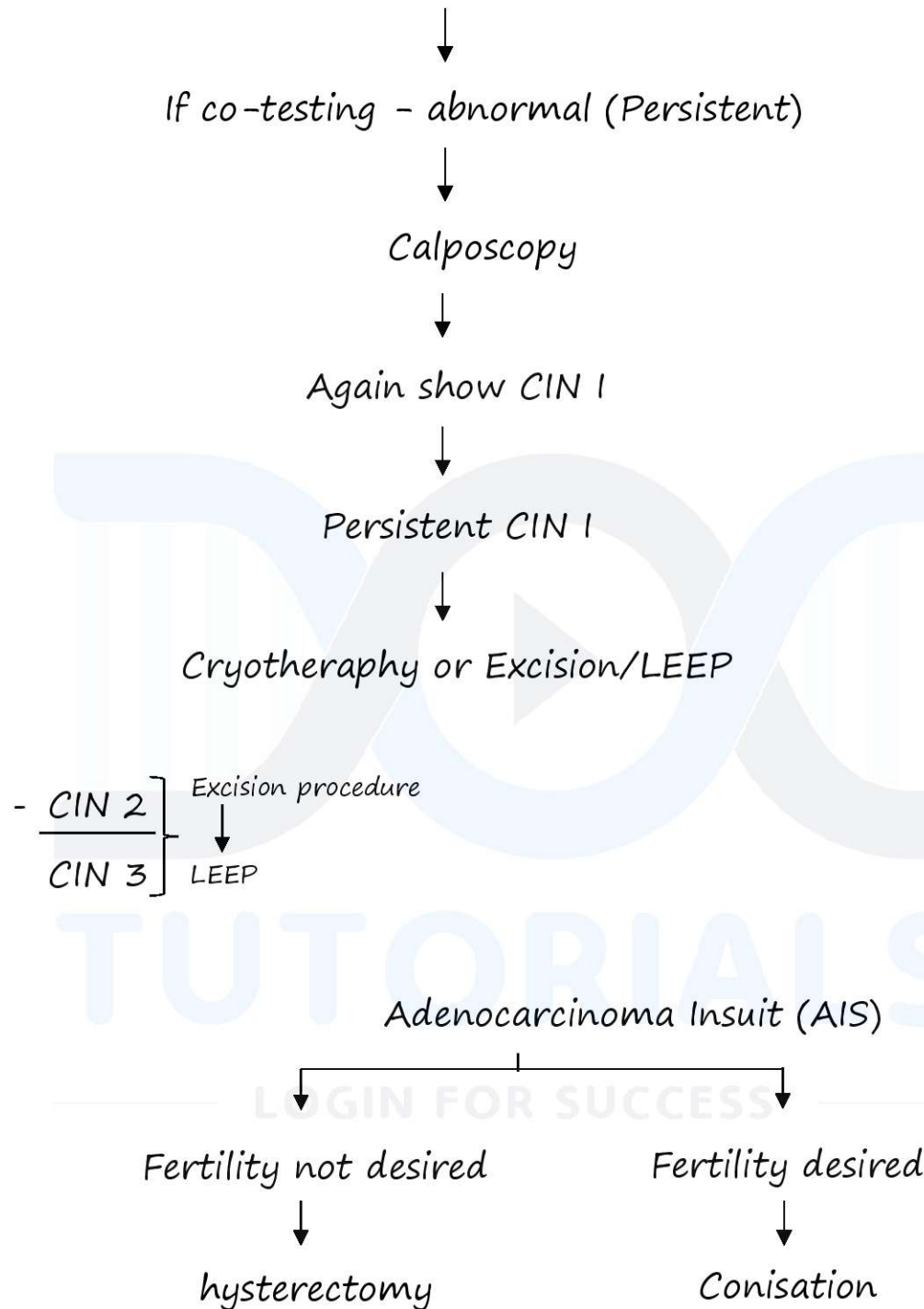
Indications for colonization

- i. Abnormal endocervical pap smear
- ii. Visible lesion extending into endocervix
- iii. CIN III
- iv. Microinvasive cervix cancer IA

Management

01:21:45

- CIN-1 → Follow cotesting every year for 2 years



Surveillance after treatment

- LSIL - Co-testing after 1yr
- ASC - H } Co-testing at 1yr, 2yr, 3yr and routine screening
- HSIL } for at least 20 years after detecting lesions

ENDOMETRIAL CANCER DIAGNOSIS STAGING

AND TREATMENT

Routes of spread

00:00:06

- Lymphatic spread
- Haematogenous spread
- Transperitoneal spread
- Direct extension
 - Pelvic nodes are first involved in endometrial cancer, they are internal iliac & external iliac and then the para aortic nodes

Staging

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00:02:25

- Ia - < 50% myometrial involvement
- Ib - ≥ 50% myometrial involvement
- II - cervical stromal involvement (does not include cervical glandular involvement)
- IIIa - serosa & adnexa involvement (Tubes & ovaries)
- IIIb - vagina & parametrium
- IIIc - C₁ - pelvic nodes involvement
- C₂ - para-aortic nodes
- IVa - bladder/bowel mucosa
- IVb - distant metastasis
 - This procedure is called as staging laparotomy
 - Firstly, the ascitic fluid should be taken for histopathology

- Peritoneal washings for histopathology
- Suspicious lesion has to be biopsied
- TAH with BSO → frozen biopsy
- Pelvic & para-aortic lymph node dissection
- Infracolic omentectomy/Bilateral peritoneal biopsy from the paracolic gutter/ diaphragm
- 3/4th of women presents at stage 1
- In this staging, type 1 hysterectomy is done, if cervical extension is present, type 3 hysterectomy (radical hysterectomy)

Treatment

00:14:07

Staging laparotomy	
Ia	Only observation is done
Ib	Adjuvant chemo radiation
II	Adjuvant chemo radiation
III	Adjuvant chemo radiation
IVa	Adjuvant chemo radiation
IVb	Inoperable, chemo only with palliative care

Poor prognostic factors.

00:16:19

- Stage
- Age – (older the pt. poorer the prognosis)
- Histological type -clear cell has bad prognosis

- Grade 3 has bad prognosis
- Myometrial invasion – more the myometrial invasion worse the prognosis
- Lymph vascular space invasion has bad prognosis
- Positive peritoneal cytology has bad prognosis
- Size of tumor – bigger the size worse the prognosis
- Expression of ER & PR – higher the expression, worse the prognosis
- Overall survival rate – 5 years – 75% for Ia disease it is 90%



UTERINE SARCOMA

- Incidence – 3% of uterine malignancies.
 - Arise from uterine muscle
(myometrium)/endometrial stroma

Classification.

00:01:10

- i. Leiomyosarcoma (60%)
- ii. Endometrial stromal sarcoma (20%)
- iii. Undifferentiated uterine sarcoma (UUS)
- iv. Adenosarcoma
- v. Perivascular epithelioid cell neoplasm (PEcoma)
- vi. Carcinosarcoma.
- vii. Malignant mixed Mullerian tumor (MMMT)

Endometrial stromal sarcoma

00:03:11

- Premenopausal women 45-50 yrs.
- AUB/Mass/Pain abdomen.
- It is divided into 2 types

Low grade ESS	High grade ESS
- <10 mitotic figure/10HPF	- > 10 mitotic figure/10HPF
- No necrosis	- Necrosis present
- Slight nuclear atypia	- Gross atypia

• Mutation in JAZ F1 – SUZ 12 - Respond to hormonal therapy - Radiotherapy only	• Mutation YWHAE – FAM - Does not respond to hormonal therapy - Combined chemoradiotherapy
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Treatment

- For both
 - Total abdominal hysterectomy with bilateral salpingo oophorectomy with pelvic chemo RT

Hormonal therapy

- Aromatase inhibitors – letrozole, anastrozole
- Progesterone

Leiomyosarcoma

00:07:31

- Median age at diagnosis is 54.
- Arises de novo or from Preexisting leiomyoma (rare) (0.13 to 0.81%)
- Associated with previous pelvic irradiation
- It is divided into 3 types
- <5 MF/10 HPF – Benign
- 5-10 MF/10HPF STUMP (smooth muscle tumors of uncertain malignant potential)

- >10 MF/HPF – malignant

Treatment

- TAH with BSO followed by chemotherapy

Pathological variants.

00:10:24

- Myxoid LMS
- Epithelioid LMS
- IV leiomyomatosis
- Benign metastasizing uterine leiomyoma
- Disseminated peritoneal leiomyoma
- Treatment – TAH with BSO and hormonal therapy

Staging ESS (ENDOMETRIAL STROMAL SARCOMA).

00:11:53

- Stage 1 – tumor limited to uterus
- 1a – endometrium
- 1b $\leq \frac{1}{2}$ myometrium
- 1c $> \frac{1}{2}$ myometrium
- Stage 2 – limited to pelvis but beyond uterus
- 2a – adnexal involvement
- 2b – other pelvic tissue
- Stage 3 – invades other abdominal tissue
- 3a – involvement of one site
- 3b – > 1 site
- 3c – pelvic/paraaortic LN

- Stage 4 -
- 4a-Bladder and rectum
- 4b-Distant metastasis

Staging LMS(LEIOMYOSARCOMA).

00:15:01

- Stage 1: tumor limited to uterus
 - 1a - < 5cm
 - 1b - > 5cm
- Stage 2
- Stage 3
- Stage 4

} Similar to ESS

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TUTORIALS

LOGIN FOR SUCCESS

CLASSIFICATION, RISK FACTORS & FAMILIAL CANCER

Ovarian Cancer:

00:00:06

Based on invasive nature

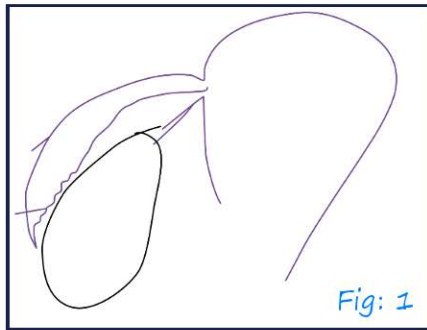
- i. Benign
- ii. Borderline → average age at diagnosis is 46 years
- iii. Malignant → epithelial ovarian cancer is 60 years

Features of Borderline tumors

- i. Epithelial hyperplasia in the form of pseudo stratification, tufting, cribriform & micropapillary architecture.
- ii. Mild nuclear atypia & mild increase in mitotic activity
- iii. Detached cell clusters
- iv. Absence of destructive stromal invasion.
 - 30 % of Ovarian tumors in post-menopausal women and 7% of those in the premenopausal are malignant.

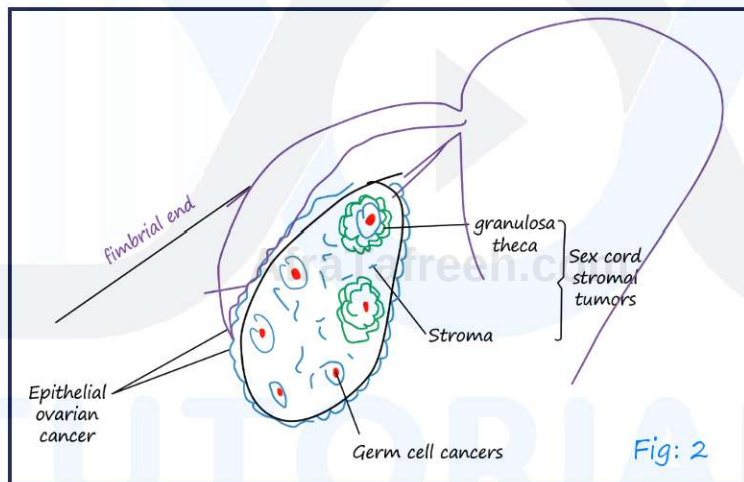
Classification Based on site:

- Most from fimbrial end



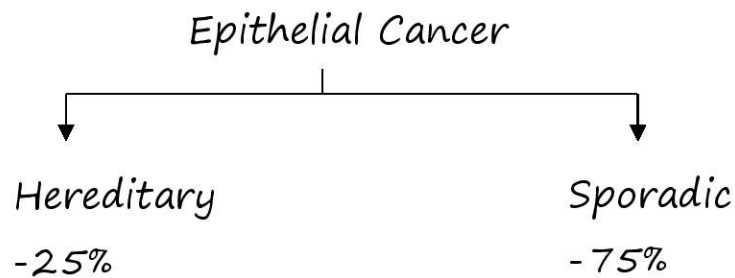
Classification based on histology

- i. Epithelial Ovarian cancer
- ii. Germ cell cancers
- iii. Sex cord stromal tumors



Epidemiology

- Most Common is epithelial ovarian cancer (60 years)
 - Mostly post-menopausal
 - Rarely young
- 2nd most common (in teenage) – germ cell
 - Fastest growing
- Least common – sex cord stromal
 - Low grade malignancies, before 20 years, 70% of tumors are germ cell in origin, 1/3rd are malignant



- Epithelial cancers are hereditary
- Germ cell cancers and sex cord stromal tumors are sporadic
- Lynch – II & BRCA 1,2 are associated with hereditary epithelial cancer
- Genetic testing should be offered to all women with Ovarian cancer
- Hereditary cancers occur 10 years earlier than sporadic cancers
- 40% hereditary cancers do not have a family history

Risk factors for Epithelial cancers

00:17:18

- More ovulation = More trauma = increases risk of ovarian cancer
 - i. Early menarche
 - ii. Late menopause
 - iii. Nulliparity
 - iv. Family history
 - v. Talc exposure to genitals

Protective Factors

00:19:36

- late menarche / Early menopause
- Multiparity
- Tubal ligation → compromised tubal vascularity → atrophy of the fallopian tube
- Combined oral contraceptives
 - Asbestos is not related to peritoneal Ca
 - Bilateral salpingo-oophorectomy is not protective for peritoneal Ca

BRCA 1 & 2

00:22:36

- BRCA 1 – Chr 17
- BRCA 2 – Chr. 13
- Autosomal dominant
- Most common malignancy – Breast cancer

	Ca Breast	Ovarian Cancer
BRCA – 1	82%	54% (50%)
BRCA – 2	82%	23% (25%)

- BRCA – 1 related Cancers occurs at an earlier age
- Commonly seen in Ashkenazi Jews

Screening

00:26:15

- 6 monthly TVS / Ca 125 levels starting at 30-35 years age

- Prophylactic BSO with total abdominal Hysterectomy (TAH can be restricted to those with increased risk of endometrial cancer) starting at 35 to 40 years for BRCA 1 and 40 to 45 for BRCA 2
- B/L mastectomy and Annual Mammogram + MRI breast alternating every 6 months
- If the woman with BRCA1/2 is not willing for surgical interventions, to prevent the cancer, combined oral contraceptives are given which decrease the risk by 50% if taken for 5 years
 - Parity reduces the risk by 30-40%

HNPCC

00:30:18

-
- Most common malignancy → colon cancer
 - Most common malignancy among women → Endometrial cancer, 10% ovarian cancer
 - Colonoscopy should be done every year starting at 20-25 years
 - Endometrial biopsy every year starting at 30 -35 years

EPITHELIAL OVARIAN CANCER CLASSIFICATION

Symptoms

00:00:06

- Ovarian carcinoma is a silent killer. Patient usually presents with symptoms during stage III/IV
- Usual symptoms are:
 - Pain abdomen
 - Mass abdomen
 - Dyspareunia
 - Urinary/fecal disturbances.
 - Hydrops tubae profluens- it is vaginal discharge seen in fallopian tube cancer.
 - USG → shows cystic/solid lesion in ovary/adnexa

Features of malignancy in USG:

00:02:47

- i. Bilaterality
- ii. Irregular borders (tumors with regular borders likely to be benign)
- iii. Septations (no septation more likely to be benign)
- iv. Internal echoes/solid components
- v. Ascites
 - There may be grossly enlarged nodes
 - May have e/o metastases and high vascularity
 - If the above findings are present in USG, it is a complex cyst.
 - Simple cyst does not have all the above findings.

Management of ovarian cyst.

00:06:00

	Pre menopausal women	Post menopausal women
Simple cyst <8cm ca 125 – Normal levels	Coc x 2 cycles ↓ Reasses	Wait and watch
Complex cyst >8cm Ca125 – level ↑ (>35 iv)	Openate staging laparotomy	Staging laparotomy

Histological types

00:09:13

- 3 gross types:
 - i. Epithelial
 - ii. Germ cell
 - iii. Sex cord stromal tumors
- Most common are epithelial tumors

Epithelial ovarian cancers – types

00:09:32

- i. Serous
- ii. Mucinous
- iii. Endometrioid
- iv. Clear cell
- v. Brenner
- vi. Mixed

vii. Undifferentiated

viii. Unclassified

- There are many types of epithelial carcinomas based on their origin (or) cells they mimic.
- Serous tumors arise from fimbrial end of fallopian tube and resemble endosalpinx.
- Mucinous tumors resemble mucin secreting GI tumors (or) adenocarcinoma of endocervix.
- Endometrioid tumors resemble proliferative endometrium
- Clear cell tumor resembles secretory/pregnant endometrium
- Brenner tumor resembles transitional cells
- Serous is the most common
- 75 – 80% belong to serous variety
- Mucinous → 5%
- Endometrioid → 10% (2nd mc)
- Brenner <1% and least common.

Serous epithelial cancers

00:13:23

Type- I	Type – II
• Borderline/low grade cancer.	• Rapidly growing/highly aggressive tumors
• Genetically stable	• unstable

• KRAS/BRAF mutation	• P ₅₃ mutations
----------------------	-----------------------------

- In serous tumors we have calcifications arranged in circle called psammoma bodies and they are due to apoptosis of tumor cells and osteo-inductive cytokines produced by macrophages.
- Serous tumors produce CA125 as tumor marker.
- Presence of psammoma bodies is a good prognosis.
- Maximum bilaterality is seen in serous tumors.
- Serous tumor is 20-40% bilateral

Mucinous tumors

00:16:35

- Contribute to 5% of epithelial cancers.
- Secretes Mucin and this tumor is biggest of ovarian tumors.
- Mucinous tumors resemble GI malignancy.
- Rupture of the tumor and release of the mucin material leads to pseudomyxoma peritonei
- Pseudomyxoma peritonei is also seen in appendiceal mucinous tumors, mucinous tumors of ovary and dermoid.
- Mucinous tumor secretes CEA and CA19-9 as tumor markers

Endometrioid tumors.

00:18:59

- 10% of epithelial ovarian cancers are Endometrioid
- Arises from endometriosis of ovary
- 15-20% of Endometrioid tumors co-exist with carcinoma endometrium
- Most common co-existing genital malignancy is Endometrioid ovarian tumor with Ca endometrium
- Variant of Endometrioid tumor – Adenofibroma

Clear cell cancer

00:21:32

- They are called Clear cell because they have cells with abundant clear (or) vacuolated cytoplasm with Hyperchromatic irregular nucleus and these cells are called hobnail cells.
- Clear cell carcinoma can also be seen vagina & cervix due to diethylstilbestrol exposure and it is always high grade.
- It may coexist with Endometrioid cancer.

Brenner tumor

00:23:19

- Resembles transitional cells
- Can co-exist with transitional all carcinoma of the bladder
- cells form groups & nests called Walthard cell nests
- Psedudomeig's syndrome



It is a combination of Ascites and right sided pleural effusion. (Called as Meigs syndrome in Fibroma)

EPITHELIAL OVARIAN CANCER STAGING AND TREATMENT

Route of spread

00:00:06

- Peritoneal spread more common than Lymphatic or hematogenous
- Lymph nodes:
 - Pelvic nodes (internal & external iliac)
 - Para aortic nodes

Prognostic factors

00:01:17

- Histological type
- Grading
- Stage
- Age of the patient
- Volume of ascites
- Residual disease after surgery

Staging

00:02:25

Surgical staging

- Procedure is known as staging laparotomy
- Done to identify:
 - Peritoneal mets
 - Nodal spread
 - To offer cytoreductive surgery (any metastasis >1cm should be removed)

Steps of staging laparotomy

- Midline vertical incision of abdomen
- ascitic fluid for cytology / peritoneal wash with 50-100ml NS for cytology
- Total Abdominal Hysterectomy with BSO
- Explore all abdominal organs in a clockwise fashion
- All Visible mets >1cm should be removed
- All Suspicious lesions → Biopsied
- Biopsy of diaphragm
- Supra colic omentectomy
- Pelvic/paraaortic lymph node dissection

Staging

- I
 - a → lesion confined to one ovary U/L
 - b → confined to both the ovaries B/L
 - C₁ → surgical spill
 - C₂ → capsule ruptured/tumor on the surface of ovary or fallopian tube
 - C₃ → Malignant ascites or peritoneal washings positive for malignancy
- II
 - a → involvement of uterus
 - b → involvement of other pelvic structures
- III
 - a₁ → pelvic/para-aortic LN involvement

- $a_1(i) < 1 \text{ cm}$
- $a_1(ii) > 1 \text{ cm}$
- $a_2 \rightarrow$ microscopic extra pelvic peritoneal metastases
- $b \rightarrow$ macroscopic extra pelvic peritoneal metastases $\leq 2 \text{ cm}$
- $c \rightarrow$ macroscopic extra pelvic peritoneal metastases $> 2 \text{ cm}$
- IV
- $a \rightarrow$ malignant pleural effusion
- $b \rightarrow$ extra abdominal met/liver & spleen parenchyma
- Serosal involvement of bowel - III
- Transmural bowel infiltration $\rightarrow$ IVB
 - Umbilical mets $\rightarrow$ IVB

Treatment

00:16:45

Stage I

Low risk	High risk
Grade 1,2	Grade 3
Intact capsule	Invading capsule
No surface excrescences	Surface excrescences present
No ascites	Ascites present
Negative peritoneal cytology	Positive peritoneal cytology
Unruptured	Pre op rupture

- Stage Ia Ib grade 1 & 2 → avoid post-op chemotherapy

Ia, Ib grade 3
 Ic
 II
 III, IV

} post-operative chemotherapy given

- Fertility preserving S_x → U/L salpingo-oophorectomy → Can be done only in stage Ia grade 1,2 after frozen section biopsy

Drugs for chemotherapy 22:47

- Ia, Ib grade 3
 - Ic, II
- } cisplatin/carboplatin +
paclitaxel } IV
- chemotherapy
- AfraTafreeh.com
- III, IV → intraperitoneal chemotherapy

+

IV chemotherapy

New drugs

- PARP inhibitors → Poly ADP Ribose Polymerase inhibitors (works well on BRCA mutant patients)
- Early stages (1,2)
 - Carboplatin Area under curve (AUC) 5 to 6
 - Paclitaxel 175 mg/m²



Every 3 weekly * 6 cycles

- Advanced stages (3,4) →

IV Day 1 – Paclitaxel 135 mg/m² IV
 + Day 2 – Paclitaxel AUC 5 IP
 IP Day 8 – Paclitaxel 60mg/m² IP



Every 3 weekly * 6 cycles

- Intraperitoneal drugs are carboplatin and paclitaxel
- Bevacizumab: 7.5 to 15mg/kg IV/IP

Platinum sensitive

- Relapse occurs later than 6 months after 1st line chemotherapy

Platinum Resistant

- Relapse within 6 months of 1st line chemotherapy

Platinum Refractory

- Disease progression while on 1st line chemo – Treat with Topotecan/Gemcitabine/Etoposide/Doxorubicin/
- Platinum sensitive – give PARP inhibitors
 - Niraparib } Works for platinum sensitive germline
 - Olaparib } BRCA and non-germline BRCA mutant patients
 - Ruca parib → For platinum sensitive & platinum resistant BRCA mutations

Survival Rates

- Stage I – 94%
- Stage II – 73%
- Stage III, IV – 28%

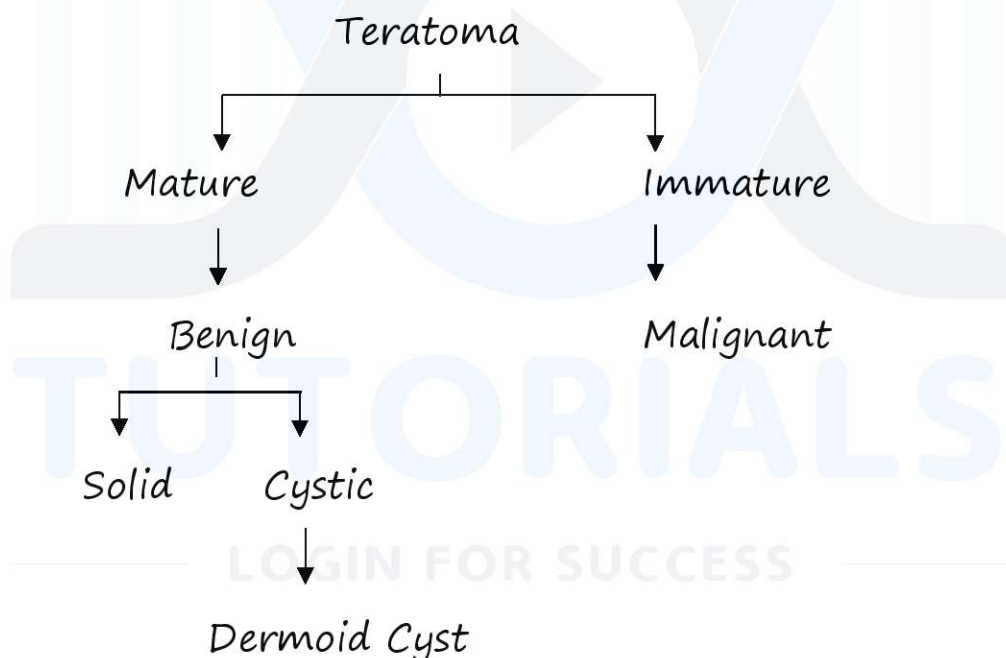


GERM CELL AND SEX CORD STROMAL TUMORS

Classification of Germ cell tumors.

00:00:15

- Primitive
 - Dysgerminoma (MC)
 - Embryonal Ca
 - Endodermal sinus tumor
 - Polyembryoma
 - Non gestational Choriocarcinoma
 - Mixed germ cell tumor.



- MC germ cell tumor $\left[\begin{array}{l} \text{Non pregnant} \\ \text{Pregnancy} \end{array} \right]$ Dermoid
- Mc germ cell cancer $\left[\begin{array}{l} \text{Non pregnant} \\ \text{Pregnancy} \end{array} \right]$ Dysgerminoma

Tumor markers

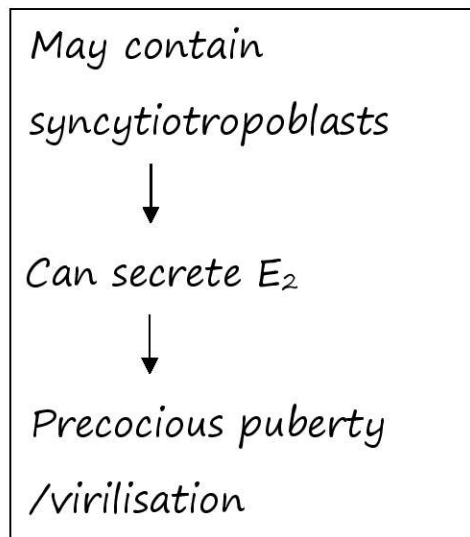
00:04:01

- Dysgerminoma – PLAP (Placental Alkaline phosphatase) and LDH (lactate dehydrogenase).
- Embryonal Ca – alphaFP and HCG
- Choriocarcinoma – HCG
- Yolk Sac tumor – alphaFP
- Teratoma – No marker
- Granulosa cell tumor – Inhibin B & AMH (Anti-Mullerian Hormone)

Dysgerminoma

00:07:01

- MC germ cell cancer
- 30-40% of all germ cancers.
- Usual age: 10-30 yrs.
- 5% of them can occur before 10 yrs.
- Both sexes are involved
- Both gonadal and extra gonadal sites (midline structures) are involved
- Usual Size: 5-15 cm.
- Have bosselated capsule with fleshy grey brown colour.
- Histology has extensive lymphocytic infiltration and has epithelioid & giant cells; Differential diagnosis is tuberculosis.



- Dysgerminoma + endodermal sinus tumor → mc coexisting germ cell tumors
- 5% of dysgerminomas develop in abnormal gonad.
- 10-15% of dysgerminomas are bilateral.
- In non-functioning gonad → Gonadoblastoma (Benign)



Develop dysgerminoma (malignant)

- Mc mode of spread → lymphatic

Treatment:

00:14:39

-
- U/L salpingo-oophorectomy for Stage Ia grade I



No adjuvant chemo

All other stages → Adjuvant chemotherapy should be given

- Stage Ib, II, III, IV → Total abdominal hysterectomy. + B/L salpingo-oophorectomy + pelvic and para-aortic LN dissection

B → Bleomycin
 E → Etoposide
 P → Cisplatin

} 3 weekly 4-6 cycles (IV)

Immature teratoma

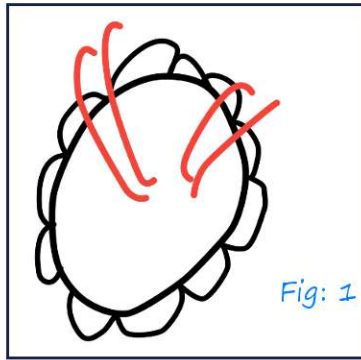
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- Rarely B/L
- 2nd mc germ cell malignancy
- Can occur in Combination with other germ cell tumors
- Secrete steroids → precocious puberty/virilisation may be seen.

Endodermal sinus tumor/yolk sac tumor:.

LOGIN FOR SUCCESS 00:18:30

- 100% U/L
- There are cystic spaces lined by flattened endothelium into which capillaries protrude like glomerular tuft. This is called schiller-duval bodies.



- Tumor marker: α FP & α_1 antitrypsin

Embryonal cancer

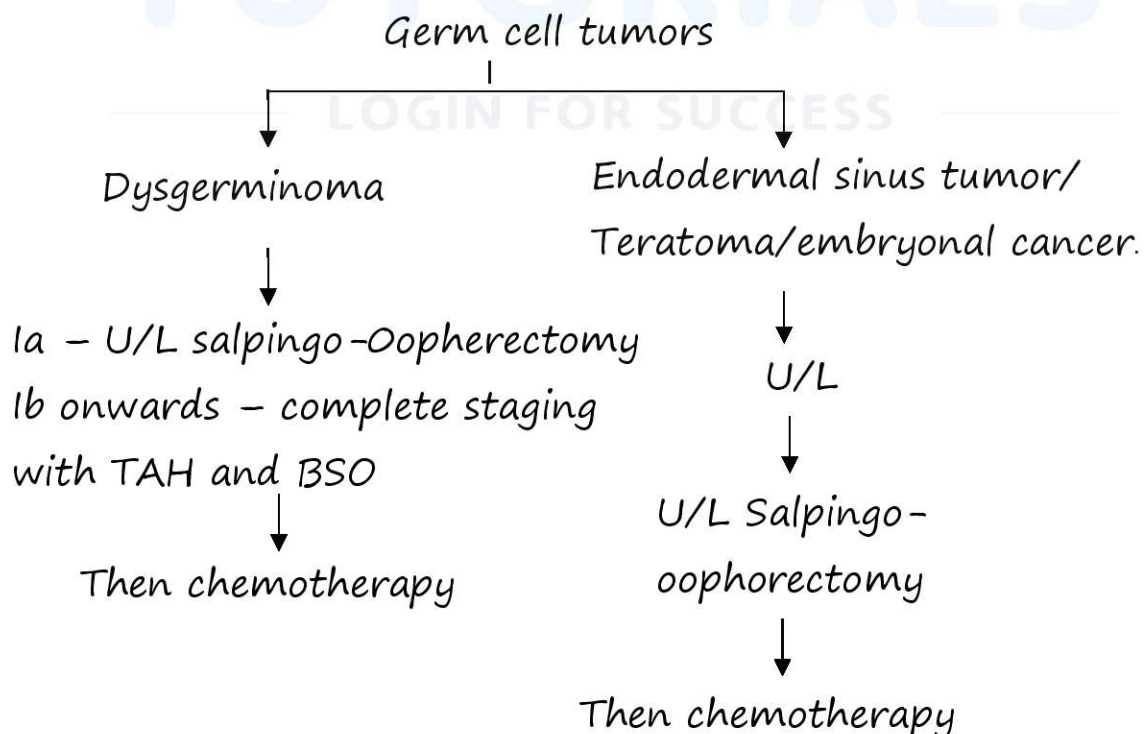
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- Seen in very young (4-28 yrs.)
- Tumor marker: α FP, HCG
- Secrete E_2 / steroid & can lead to precocious puberty/ AUB

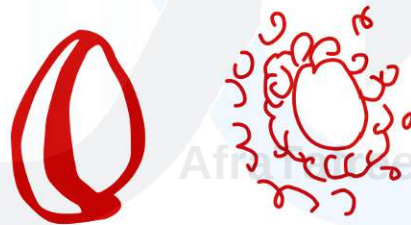
Choriocarcinoma

00:20:31

- Can secrete steroids
- Lead to precocious puberty



- Granulosa cell tumor:
 - 70% of sex cord stromal tumors are granulosa cell tumor
 - There are 2 types
 - i. Adult – 95% → Associated with FOXL₂ gene mutation seen.
 - ii. Juvenile – 5%
 - Rarely B/L
 - Coffee bean nuclei seen



Carl Exner bodies – cluster of cell around central cavity

- They secrete
 - Estrogen → causes precocious puberty/AUB
 - Androgen → virilisation
 - Inhibin B } Tumor markers
 - AMH → }
- Associated with estrogen excess and hence causes:
 - Endometrial hyperplasia – 25-50%
 - Endometrial cancer – 5%

Treatment

- Young – U/L salpingo-oophorectomy → chemo
- Post-menopausal – surgical staging & TAH and BSO ± chemo

Sertoli Leydig cell tumors

00:30:11

- Involves women <40 years
- Secretes androgen → leads to virilisation/hirsutism
- DICER 1 gene mutation is seen
- Maximum U/L
- < 1% are B/L

Treatment

- Young → U/L salpingo oophorectomy → followed chemotherapy

Metastatic ovarian cancers.

00:32:28

Krukenberg tumor

- Mc metastatic ovarian cancer
- Mc site for primary is Stomach
- Other sites
 - Colon
 - Appendix
 - Breast/ biliary tract/ cervix
 - Bladder
- Usually B/L

- Sometimes, they secrete thyroxine and are called *Struma-ovarii*
- Mucin filled cells are seen called as signet ring cells.
- Retain the shape of ovary



VULVAL CANCER -I

- Most common anogenital cancer in women > 70y rs
- Predominantly seen in post-menopausal women

Etiology/Risk Factors.

00:02:42

- i. Human papilloma virus [HPV]
- ii. CIN – Cervical intraepithelial neoplasia
 - VIN – Vulval intraepithelial neoplasia
- iii. Cervical cancer
- iv. Lichen sclerosis – inflammatory condition of vulva
- v. Smoking
- vi. Alcohol
- vii. Obesity
- viii. Immunosuppression
 - Now there is increasing incidence of vulval cancer due to chronic inflammatory conditions
 - Incidence of vulval cancer due to HPV is decreasing because of HPV vaccine

Types.

00:04:35

1. Squamous cell carcinoma
 - Most common
 - 90%
2. Malignant melanoma
 - 2nd most common, 2.5%

3. Basal cell carcinoma – 2-3%

4. Paget's disease

5. Bartholin's type

6. Adenocarcinoma – arising from sweat gland,
Skene's gland or Bartholin's gland.

7. Ewing's sarcoma

8. Rhabdomyosarcoma

9. Leiomyosarcoma

10. Epithelioid sarcoma

11. Neuroendocrine tumors involving vulva

12. Lymphoma involving vulva

<1

Squamous Cell Carcinoma.

00:08:01

- Types

i. Warty/basaloid type

ii. Simplex/keratinizing/differentiated type

Basaloid/Warty	Keratinizing/Simplex
- 40% - Multifocal - Young patient - HPV associated - Usual VIN - Immunosuppression - Smoking	- 60% - Unifocal - Older/postmenopausal - Lichen sclerosis association - Differentiated type of VIN

- Cancer Cervix
 - HPV 16 → Most common
 - HPV 18, 45 – next common
 - HPV 31, 33
 - Cancer vulva – HPV 16 – most common
 - HPV 18, 33
 - Around 1-2% vulval cancer associated with other malignancies
- ↓
- Malignancies associated with smoking
 - Ca. Larynx
 - Ca. Pharynx
 - Ca. Lung
 - Malignancies associated with HPV
 - Anal Cancer
 - Cervical cancer
 - Vaginal cancer
 - Laryngeal cancer

Symptoms of Vulval Cancer.

00:14:14

- i. Pruritus – Earliest clinical manifestation
 - ii. Ulcer /mass or lump → most common presenting complaint
 - iii. Bleeding /discharge/pain
 - Most common site: Labia majora
 - 2nd most common site: Labia minora
- } 60%

- Other sites
 - Clitoris – 15%
 - Urethral orifice
 - Perineal skin – 10%

Mode of spread.

00:17:31

- Lymphatic spread – most common
- Hematogenous
- Direct
- Sentinel lymph node

Superficial inguinal lymph nodes



Deep inguinal lymph nodes



Femoral nodes



External iliac nodes

More the lesion towards the midline within 2cm

more likely to involve bilateral lymph nodes

Lesions involving the clitoris/any midline structure



Can directly involve the deep inguinal nodes without
superficial inguinal lymph nodes

NATURAL METHODS

Contraception

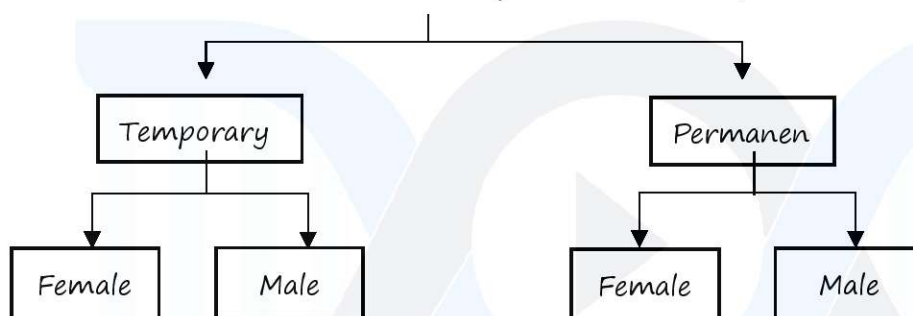
00:00:19

- It is a technique to prevent unwanted pregnancies, either temporarily or permanently.

Classification

00:00:36

- Classification of contraception techniques



Temporary female technique - Types

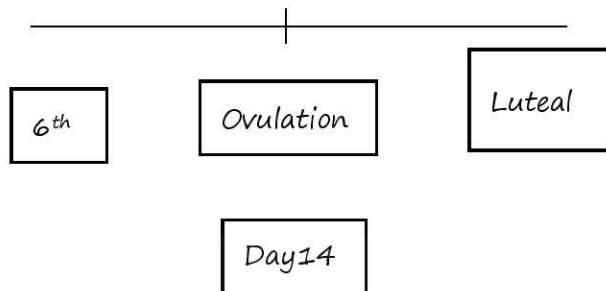
- Natural
- Barrier
- Intra uterine devices
- Hormonal techniques
- Temporary female technique is the most commonly used technique (temporary)

Natural methods:

- Rhythm/calendar method (Ogino knaves' theory)
- Standard Days Method (Ritumala method in India)
- Billing's/cervical Mucus Method
- Basal Body Temperature Method
- Symptothermal method

- Withdrawal/coitus interruptus
- Lactational Amenorrhea (LAM)

Rhythm/Calendar method



- Normal duration of menstrual cycle – 28 days
- The luteal phase is always constant
- For a 28-day cycle ovulation occurs at day 14
- For a 30-day cycle ovulation occurs at day 16
- The ovulation can occur between Day 7 to Day 21, hence to avoid pregnancy, coitus should be avoided between these days

Standard days method (ritumala method in India)

- It was used in women with regular periods, but it is not in practice now

Billing's/cervical mucus method

- Under the influence of estrogen, cervical mucus becomes thin and watery in the first half of menstrual cycle whereas under the influence of progesterone the cervical mucus becomes thick and scanty in the second half of the menstrual cycle

- Before LH surge, estrogen reaches its peak making the cervical mucus thin and favourable for penetration of sperms. Coitus should be avoided around this period.

Basal body temperature

- In this technique woman checks her temperature early morning around the anticipated time of ovulation, increase in temperature by 0.4 to 0.8 degree C indicates ovulation.

Symptothermal method

- It is a combination of a cervical mucus method & basal body temperature method

Coitus interrupts/withdrawal

- In this method, the male partner withdraws the penis before ejaculation

Lactational amenorrhea

- Amenorrhea and anovulation associated with lactation due to increased prolactin protects against pregnancy

Pre requisites

- Feeds should be initiated soon after delivery
- Exclusive breast feeding
- At least one feed at night
- Frequency is more important than duration
- Failure rate is 1-2 per hundred women years

MCQs on LAM

- Most commonly adopted natural FP method
- Natural fp method with least failure rate
- After how many weeks does ovulation resume in a non-lactating mother-6weeks
- In lactating mother-6months

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TUTORIALS

LOGIN FOR SUCCESS

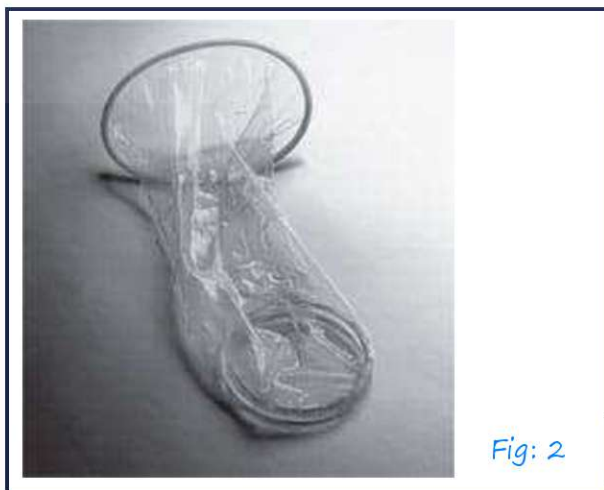
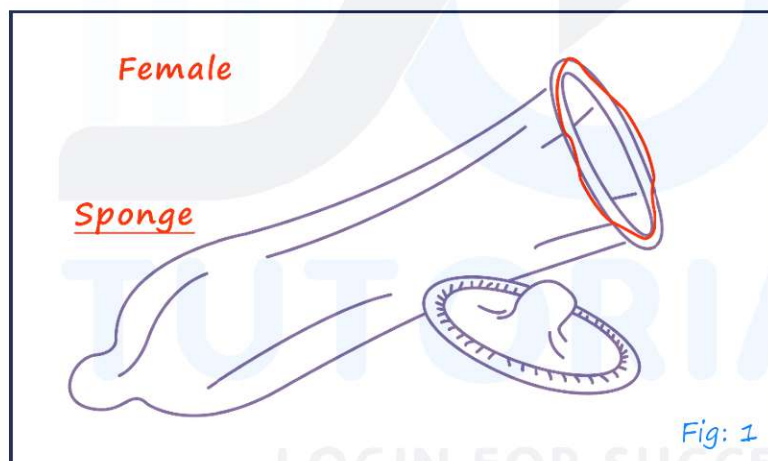
BARRIER METHODS

- Condoms
- Diaphragm
- TODAY/ cervical sponge

Condoms

00:00:32

- It is of 2 types
 - Male (fig 1)
 - Female (fig 2)



- Condoms are made up of polyurethane

- Condoms prevent again STD's/HIV

Non contraceptives benefit of Male Condoms:

- It can be used during post-partum haemorrhage for uterine tamponade
- It can be used to cover a transvaginal probe
- Used in Vaginoplasty
- Used in treating Antisperm antibodies

Femshield female condom – it is known as “FEM” or “Femidom”

- Female condom is longer than male condom, 15-17cm long
- Made of polyurethane
- Loose fitting
- Highly protective against HIV & STD
- Can be removed immediately after intercourse

Advantages of using a female condom is

- It is coital independent
- It can be worn in puerperal period

Disadvantages

- Failure rate 5-15/HWY
- Expensive
- It can be reused only once

Diaphragm

00:07:48



Cervical Cap



Diaphragm

- It is made of rubber with a metal rim on the margins
- It cannot be removed immediately after intercourse, It has to be left in place for atleast 6-8 hours after coitus
- It Cannot be used in the puerperal period
- Dreadful complication associated with the use of diaphragm is toxic shock syndrome
- It Can be reused for one year.

Contraindications for use of diaphragm

- Genital prolapses
- Cystocele/ rectocele
- Retroverted uterus
- Genital Fistulas
- Eroded/lacerated cervix
- Recurrent UTI

Failure rate:10-30 /HWY

Cervical sponge/TODAY

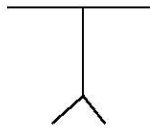
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- It is made of polyurethane
- It contains spermicide – nonoxynol-9
- It is effective for 24 hours
- The failure rate is 9-30/HWY
- It can cause toxic shock syndrome (TSS)

IUCDParts and composition of IUCD

00:01:02



- IUCD has a vertical and a horizontal arm and also a thread
- Some devices have copper only on the vertical arm while certain other devices have copper wound on the horizontal arm as well
- The filament component of IUCD is usually made by polyethylene
- Copper is usually wound on filament of IUCD but a device called NOVA T has silver as well as copper wound around it
- Some IUCD's are progesterone impregnated
- The thread is made of Nylon
- Horizontal arm of IUCD is placed in the fundus
- Position of IUCD is confirmed by visualising the thread in vaginal examination
- IUCD is also located in X ray by making it radio-opaque by coating it with BaSO_4
- The thread also enables easy removal of the IUCD
- The junction of horizontal arm with vertical arm has a spring which enables the IUCD to either open or fold its arms/wings
- Memory time of IUCD is the time taken for the arms to open from folded position
- Memory time is usually 2 minutes

Generations of IUCD

00:04:43

- There are 4 generations
- 1st generation
 - Inert e.g. Lippes loop, saf T coil, china coil
- 2nd generation
 - Copper containing devices
 - e.g. Cu T 380 A, Cu T 200, multiload 250, 375
- 3rd generation
 - Hormone containing devices
 - They contain Levonorgestrel

- e.g. Mirena, progestasert
- 4th generation
 - It is either a frameless device or a combined copper hormone device
 - eg. Gynefix (frameless IUCD in development)

Cu T 380-A and Multiload 375

00:08:34

- **Cu T 380-A**
 - Also called 'PARAGARD'
 - It is T shaped device
 - 380 mm² of surface is covered with copper
 - It has copper on both horizontal and vertical arm
 - It has a lifespan of 10 years
 - It has BaSO₄ to make it radio opaque
 - It has plunger for easy insertion
 - Made of polyethylene
- **Multiload 375**
 - It is umbrella shaped and is serrated
 - Does not have BaSO₄
 - No plunger presents
 - Made of polypropylene
 - It has a lifespan of 5 years
 - 375 mm² of surface area is covered with copper

Hormone Containing Devices

00:14:55

- **Progestasert**
 - Contains 38mg LNG
 - Releases 65 microgram/day
 - Acts on ovary in 40%
 - It has a lifespan of 1 year
- **Mirena**
 - Contains 52mg LNG
 - 20 mcg/day is released
 - No systemic action
 - No action on pituitary/Hypothalamus
 - Acts only on uterus
 - It has a lifespan of 5 years
- Incidence of ectopic pregnancy is higher with progestasert

- Progesterone decreases tubal motility and increases the chance of ectopic pregnancy
- Progesterone only contraception leads to ectopic pregnancy
- The failure rate of Mirena is 0.1 to 0.4 per hundred-woman years

Copper containing devices and their lifespans

00:19:10

Device	Cu released/day (mcg)	Life span
Cu T 200	50 mcg	3 years
Cu T 200B	50 mcg	4 years
Multiload 250	60-100 mcg	3 years
Multiload 375	30 mcg	5 years
Cu T 380 A	30 mcg	10 years

- Cu T 380 A has 66mm² surface of copper in horizontal arm (33mm² in each half) and 314mm² surface of copper in vertical arm.



Fig: 1

Mechanism of action of copper containing devices

00:22:36

- Forms biological film containing fibrin, phagocytic cells, proteolytic enzymes all of which inhibit mobility of sperms
- Copper is proinflammatory which increases PG production and leucocyte infiltration
- Copper also causes enzymatic and metabolic changes in endometrium making it unsuitable for implantation

Timing of insertion of IUCD

00:25:06

- Post placental – within 24hrs of delivery
- Post abortion – any time after abortion

- Post menstrual – ideally at the end of menses
- Intra caesarean

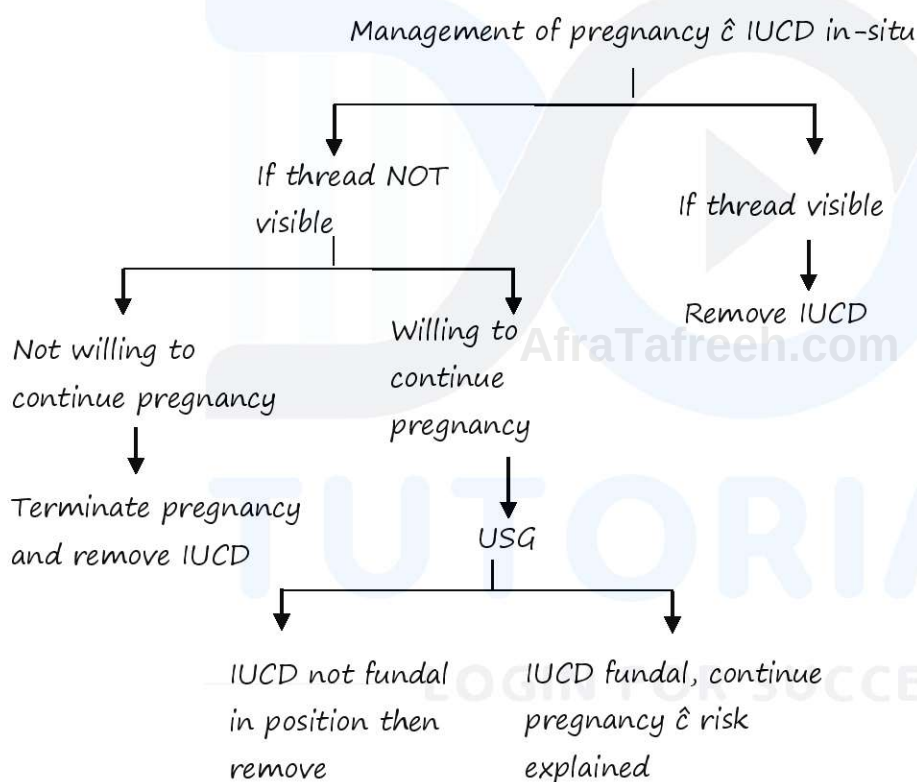
Techniques of insertion

- Withdrawal technique: plunger is used to insert IUCD into the uterus and arms are released on withdrawal of plunger

Pregnancy with IUCD

00:27:49

- Copper containing IUCD's are not teratogenic
- LNG containing IUCD's are androgenic
- IUCD's increases the chance of infection so it is ideal to have it removed in the 1st trimester



Missing thread

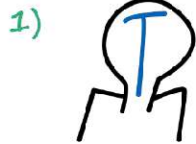
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Possibilities:

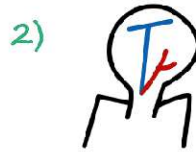
- The thread has detached from IUCD
- The thread is inside the uterus
- The IUCD has perforated
- The IUCD has been expelled from uterus

Missing IUCD

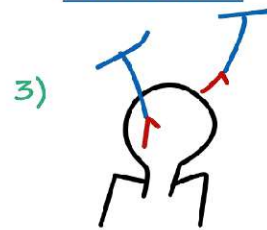
Thread detached



Thread inside uterus



Perforation



Expulsion



- Investigations
 - 1st investigation → transvaginal ultrasound
 - 2nd investigation → X-ray either with uterine sound or newly inserted IUCD
 - Next investigation → Laparotomy to remove perforated IUCD
- Detached thread and thread inside uterus are detected by transvaginal ultrasound
- Perforated IUCD is detected by X-ray
- If IUCD is present inside the uterus hysteroscopy guided removal is done
- Laparotomy is preferred over laparoscopy for removal of perforated IUCD because there may be dense adhesions and synechiae
- Failure of Cu containing IUCD's is around 1%

Complications

00:38:43

- Expulsion
- Perforation
- Infection
- Pain
- Bleeding
- Vasovagal shock
- Cramps
- Infection by actinomyces
 - Most common side effect → Bleeding
 - 2nd most common side effect → pain
 - Most common cause of Removal → pain
 - Most serious complication → perforation

- Expulsion rate of IUCD → 5%
- Perforation rate of IUCD → 1-2%

Contraindications

00:41:00

- Pregnancy/Amenorrhea
- Septic abortion
- Unexplained vaginal bleeding
- Gestational trophoblastic diseases
- Endometrial cancer
- Cervical cancer
- Fibroids
- Uterine anomaly
- Current PID
- Breast cancer
- Copper allergy
- Wilson's disease

Not a contraindication

- Nullipara
- Heart disease
- Scarred Uterus
- HIV positive

Newer IUCD

00:43:53

- Fibroplant
 - LNG containing IUCD
 - Releases 14mcg/day
- Gynefix
 - Frameless IUCD
 - Has copper cylinders tied to string
 - Requires hysteroscopy for insertion

Non contraceptive uses of copper devices

00:45:08

- Emergency contraception
- To Prevent adhesions after adhesiolysis in Asherman's
- After Septal resection in septate uterus

- Abnormal or dysfunctional uterine bleeding
- Endometrial hyperplasia
- Endometrial cancer stage Ia
- To prevent oestrogen induced endometrial hyperplasia in HRT
- During tamoxifen therapy to prevent endometrial hyperplasia
- Fibroids
- Endometriosis
- Adenomyosis
- PMS



HORMONAL METHODS

Classification

00:00:18

- i. Combined Contraception: - Estrogen + Progesterone
- ii. Progesterone only contraception

Combined

- Oral pills (COC)
- Injections
- Transdermal skin patches
- Vaginal rings

Progesterone only

- POP (oral pills)/ (Minipills)
- Injections
- Implants
- IUD
- Vaginal rings

Generations of progesterone

00:02:11

1 st generation – Norethindrone /Lynesternal Nor ethynodrel	Androgenic Property - Moderate
2 nd generation – Norgestrel, Levonorgestrel	Androgenic Property -Highest, decrease HDL, increase LDL
3 rd generation – Desogestrel, Norgestimate, Gestodene (Potent Anovulatory drugs) (lipid friendly) (increase VTE)	Androgenic Property -Lowest
4 th generation – Drospirinone, Dienogest – Cyproterone acetate	Anti- mineralocorticoid
MALA-N belongs to 2 nd generation	

Preparation of Combined Contraception

- Oral → MALA –N₁ MALA-D (EE₂ + LNG) -21 tabs
plus ferrous fumarate – 7 tablets
- Injection
- Vaginal Rings
- Skin patches

Vaginal Rings

00:10:06

- Nuva ring – Ethinyl Estradiol + Etonorgestrel
(Derivative of Desogestrel)
- Increase Venous thromboembolism
- Anovulatory effect
- lipid friendly

Injections

00:11:36

- i. Cyclofem – estradiol Cypionate + DMPA (Depot medroxy Progesterone acetate)
- ii. Mesigyna /Lunelle – Estradiol valerate + NET – EN
(norethisterone enanthate)
- iii. Marvelon – Ethinyl Estradiol + Desogestrel
- iv. Femovan – Ethinyl Estradiol + gestodene.

Skin Patch

00:13:47

- Orthoevra – EE2 + Norelgestromin

Classification of COC

00:14:45

- i. According to change of dose in different phases of cycle
- ii. No. of days at a stretch before pill free period.
- iii. According to type of progestin
- iv. According to type of estrogen
- v. According to dose of estrogen

- According to change of Dose in different phases of cycle (Phasic pills)
 - Monophasic: - In a single strip of COC pill we have 28 tablets out of which 21 will be hormonal containing tablet & 7 tablets will be iron tablet
 - MALA - N - 30 micrograms of EE_2 + 0.15mg LNG and the amount of estrogen and progesterone will not vary with each pill rather all the 21 tablets will be having the same levels of estrogen & progesterone.
 - Femilon - 20 microgram EE_2 + 0.15mg Desogestrel
 - Novelon - 30 micrograms EE_2 + 0.15mg LNG

Biphasic pill

- within the given single packet of 21 tablets, 3 tablets. 3 types of tablets will be present each having different dose of estrogen and progesterone.
 - 30 micrograms of EE_2 + 0.15mg of LNG (D_1 - Day6)
 - 40 micrograms of EE_2 + 75mg LNG (D_7 - Day₁₁)
 - 30 micrograms of EE_2 + 125mg LNG (Day12 - 21)

According to Number of days at a stretch

Number of days at a stretch	D1 of cycle
1- 28-tab pack → 21 hormone tabs + 7 /Iron tablet	(21+7) =28days
2 - 24 hormone + 4 iron tablets	(24+4) 28 days
3. Seasonable/Extended pill 84 hormone tablets /start at D1 /used in Endometriosis/ PCOS	

Types of Estrogen

00:23:04

- Ethinyl Estradiol
- New preparations like: - Mestranol – Estradiol Valeate

Types of Progestin

00:23:49

- 4 generations

Dose of Estrogen & Progestin

- 50 micrograms of EE_2 + 0.15mg LNG → used before
- not used now as higher the dose of Estrogen more the risk of thromboembolism

- 50 micrograms → 30 micrograms → 20 micrograms → 10mg pill



High dose pill Low dose pill minimum required

- Low dose has less complications, risk of VTE is less
- Disadvantage → less efficacy and increased risk of break through bleeding

When to start a combined pill

- Non lactating /Non puerperal → D₁ of menstrual cycle/ Sunday start.
- Puerperal /Lactating → Decrease Quantity of breast milk. start after 6 months for COC and after 3 weeks for POP
- Post abortal - start anytime /ideally within 7 days of evacuation
- Non - lactating mother → start after 3 weeks post-partum

How to take

- one daily, preferably at night time and at same time every day

Contraceptives provided through Government of India

- Combined pill -MALA -N
- Emergency pill - ezy pill (1.5mg LNG)
- IUD - CUT 380A, multi load 375

- Injection DMPA – Antana
- Male condoms – Nirodh
- Non hormonal contraceptive drug- SERM –
Centchroman / Ormeloxifene

Mechanism of Action COC

00:35:07

- Estrogen inhibits FSH/GnRH – Anovulatory
- Progesterone prevents LH surge, makes endometrium thin – Implantation cannot occur and Cervical mucus becomes thick
- Decrease tubal motility – Progesterone

Non-Contraceptive benefits

00:37:24

- Ovulatory pain
- Menorrhagia/AUB
- Dysmenorrhea
- Premenstrual syndrome
- Irregular periods
- hyper androgenism / PCOS
- Endometriosis
- Premenopausal hot flushes
- Decrease osteoarthritis
- Improves anemia
- Prevents ectopic pregnancy
- Prevents PID
- Benign breast disease

- Functional ovarian cyst
- Decrease Risk of Rheumatoid arthritis
- Decrease bleeding in fibroids

Malignancies & COC

00:40:48

- It decreases the risk of
 - endometrial cancer
 - ovarian cancer
 - Colon cancer
 - Choriocarcinoma

Increases the risk of

- i. Cervical Cancer – adeno Carcinoma
- ii. Hepatocellular – adenoma
- iii. Breast Cancer (mild increase)
- iv. Pituitary adenoma.
 - no increase in liver cancer with COC

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Adverse effects

00:42:49

- Estrogen: -
 - i. Nausea /Vomiting
 - ii. Loss of appetite
 - iii. Chloasma
 - iv. Headache /Migraine
 - v. VTE
 - vi. MI / Stroke

vii. Cholestasis.

Progesterone

- i. Break through bleeding
- ii. Weight gain
- iii. Edema
- iv. Depression
- v. Decreased libido
- vi. Acne
- vii. Greasy Scalp

Metabolic effects

- Decrease glucose tolerance
- Estrogen / 3rd generation progesterone → lipid friendly
- 1st, 2nd generation progesterone → Dyslipidemia
 - Estrogen increases SHBG, Angiotensinogen, clotting factors

Contra indications

00:46:24

Absolute

- Past history of VTE/ATE
- Liver cell failure / liver cancer
- CAD/CVA
- Migraine with aura
- Diabetes Mellitus with Vascular disease

- known / suspected breast Cancer
- undiagnosed Vaginal bleeding
- Suspected pregnancy
- Age >35 with smoking
- Severe hypercholesterolemia
- Uncontrolled Hypertension
- Breast feeding and less than 3 weeks' post-partum

Relative

- Migraine without aura
- controlled Hypertension
- Gestational Diabetes Mellitus
- Seizure disorder
- Diabetes Mellitus
- Obstructive Jaundice in pregnancy
- Sickle cell disease
- Gall bladder disease
- Mitral valve prolapse
- SLE
- Hyperlipidemia
- Smoking with age <35
- Hepatic disease

Missed pill management

- If 1 pill is missed → Take the missed pill as soon as she remembers it.
- Resume the schedule

- Backup is not required
- If two pill missed → If it is week ½ → take 2 pills daily for 2 days and finish the pack. Backup required for 7 days
- If two pill missed → If it is week 3 → start a new pack backup is required for 7 days.
- If 3 pill missed → start new pack. backup required for 7 days

Progesterone only pills

- LNG containing pills – 2nd generation – Also called as minipill.
- Cerozette – desogestrel only pill – 3rd generation pill,

Depot injections

- DMPA – Depo medroxy progesterone acetate (150mg/3 monthly)
- NETN – Nor ethisterone enanthate (200mg /2 monthly)

Subdermal implants

- Nor plant 1 – LNG containing – 6 rods → each 35mg LNG
- Nor plant 11 – LNG containing – 2 rods → each with 70mg of LNG with daily release of 50 micrograms with 3 to 5 years life span.

- Implanon – Desogestrel containing, it has 67mg desogestrel /daily release is 30 micrograms.
- Capronor – LNG containing – biodegradable

When should you insert an implant

- First day of menses
- Within 5 days of abortion
- 3 weeks after delivery

Technique and Site

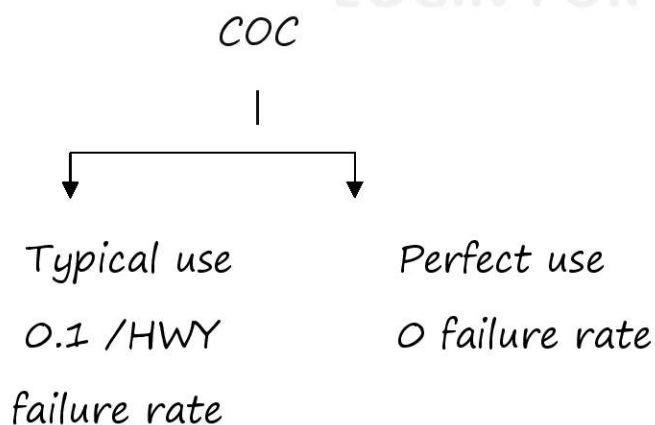
- Under local anesthesia
- Medial aspect of upper arm.

Advantage of POP	Disadvantage of POP
- Diabetes	- Lipid abnormality
- Hypertension	- Porphyria
- Sickle cell anemia	- Ectopic pregnancy histon
- Lactating mothers	- Ovarian cyst
- Women more than 35 years	- Breast malignancy
- Smokers	- Unexplained vaginal bleeding
- Contraindications of POP	
- Previous ectopics	

- ovarian cyst	
- Breast and genital malignancy	
- Active liver and arterial disease	
- Porphyria	
- Liver tumors	

Failure rates

- Natural - > 25 /HWY
- LAM - 1-2 /HWY
- Mirexa - 0.1 - 0.4 /HWY
- Condom - 5-15/ HWY
- Diaphragm - 10-30/HWY
 - Pop - Cerozette - 0.21/HWY
 - Depot injections - 0.4 to 0.6 /Hwy
 - Minipill (LNG) - 1-2/HMY.



DEFINITION, CAUSES AND STAGING

Supports of uterus

00:00:31

Mechanical support

- Uterine axis
- Angle of ante-version and anti-flexion
 - Angle between uterus and cervix is called flexion and is about 120°
 - Angle between cervix and vagina is called version and is about 90°

Muscular support

- Pelvic diaphragm (Levator ani)
- Perineal body
- Urogenital diaphragm

Ligamentous support

- Cardinal/mackenrodt's/Transverse cervical ligaments
- Uterosacral ligaments
- Pub cervical ligaments
- Most important support of the uterus is the cardinal/mackenrodt's/transverse cervical ligament
- Transverse cervical ligament also support upper vagina along with uterus

De Lancey's 3 level support system for vagina

00:07:40

- Level I → Cardinal and uterosacral ligaments
- Level II → Endopelvic fascia (formed by vesicovaginal and rectovaginal fascia)
- Level III → perineal body, perineal membrane and perineal muscle
 - Perineal body
 - Perineal membranes
 - Perineal muscles including levator ani
- Level I defect leads to vault prolapse/Enterocoele
- Level II defect leads to cystocele/Rectocoele
- Level III defect leads to Gaping introitus and urethrocele

Risk factors for prolapse

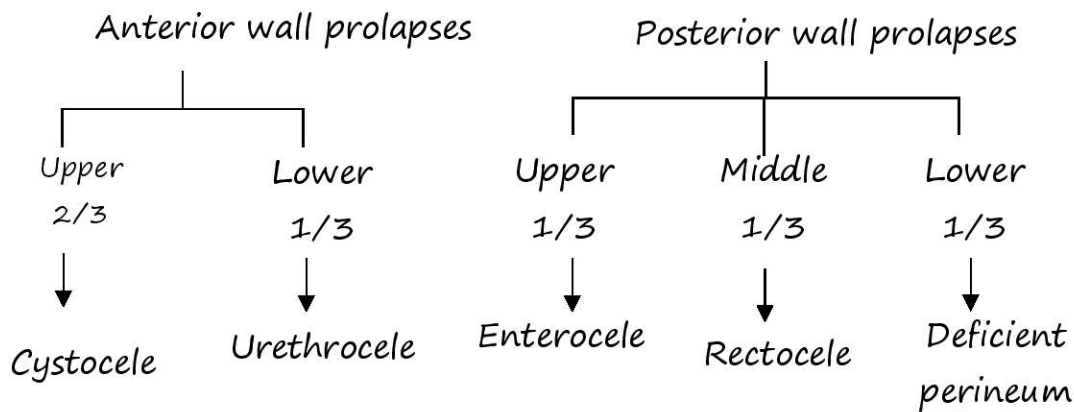
00:14:47

- Weakening of pelvic supports
 - Pregnancy/Delivery
 - Multiparity
 - Age
 - Menopause
 - Congenital defect
 - Hysterectomy
- Chronic increase in intra-abdominal pressure
 - Smoking

- Constipation
- Chronic lung disease
- Obesity
- Occupational
- Obstetric factor
 - Vaginal delivery
 - Large foetus
 - Instrumental delivery
 - Prolonged second stage
 - Premature bearing down
 - Multiparity

Anatomical classification of prolapse 00:16:47

- In conventional classification reference point is Ischial spine
- Normally :- External OS is at the level of ischial spine
- 1st degree prolapse – External OS is below Ischial spine but above hymen
- 2nd degree prolapse – External OS at level of hymen
- 3rd degree prolapse – External OS outside the hymen
- Proceidentia – External OS of cervix is outside the hymen



POP-Q (Pelvic Organ Prolapse – Quantification)

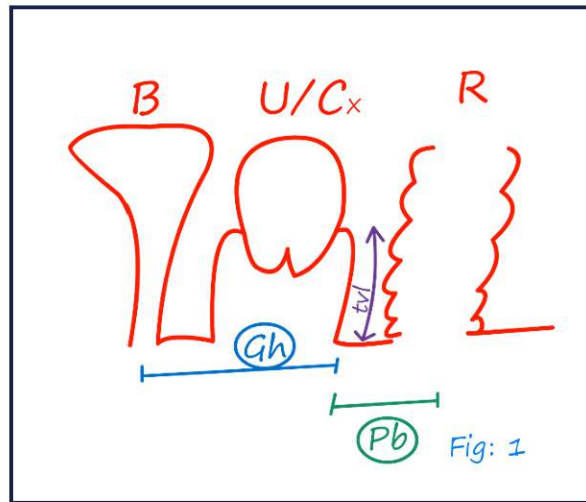
- Reference point – Hymen
- Any point above Hymen is given positive value and any point below is given a negative value
- Grid

Aa	Ba	C
Gh	Pb	TvL
Ap	Bp	D

- Genital hiatus (Gh) → it is the distance between the middle of urethral orifice and posterior hymen
- Perineal body (Pb) → distance between posterior hymen and middle of anal opening
- Total vaginal length (TVL) → distance between hymen and posterior fornix
- Aa → a fixed point on anterior wall of vagina which is 3cm proximal to hymen

- Ba → distal most point on the anterior wall of vagina
- Ap → fixed point on posterior wall 3cm proximal to hymen
- Bp → distal most point on the posterior wall of vagina
- When no prolapse → Aa and Ap on same level and Ba and Bp are on same level
- In normal anatomical position, value of Aa is -3
- In complete prolapse, value of Aa → +3
- So range of Aa → -3 to +3
- Similarly range of Ap → -3 to +3
- Range of Ba/Bp → -3 to TVL
- 'C' is the distance between tip of the cervix and hymen
- 'D' is the distance between attachment of uterosacral ligaments and hymen
- There's no point D in hysterectomised patients as the uterosacral ligaments are absent
- POP grid of complete prolapse in hysterectomised patient whose TVL is 8cm is given below:

+3	+8	8
4	5	8
+3	+8	X



POP-Q staging

00:46:52

- Stage 0 – no prolapse
- Stage 1 – all points ≥ 1 cm above the hymen
- Stage 2 – all points within 1 cm from hymen
- Stage 3 – all points are 1 cm below the hymen
- Stage 4 – complete eversion of vagina

Differential diagnosis of prolapse

00:50:13

- Cervical polyp
- Endometrial polyp
- Infravaginal elongation of cervix
- Gartner's cyst
- Inversion of uterus

MANAGEMENT

Decubitus Ulcers

00:00:17

- Due to venus congestion (alteration of anatomy of veins around the area)
- Rarely malignant

Management of Decubitus Ulcer

- Reduce the prolapse
- Pack the prolapse
- Elevate limbs
- Estrogen cream for postmenopausal women

Management of Prolapse

00:01:12

- i. Young nulliparous lady (congenital weakness)
- ii. Young multipara's lady
- iii. Old women fit for surgery
- iv. Old women unfit for surgery
- v. pregnancy

For a pregnant woman

- i. Pessary until 16weeks (remove at 16weeks)
- ii. Until 2weeks postpartum, the prolapse does not recur
- iii. Insert pessary after 2weeks postpartum until 6weeks postpartum after which remove the pessary and offer definitive treatment

Pessary → Insert as soon as prolapse becomes symptomatic



Until 16 weeks



Remove pessary until 2 weeks postpartum



Re insert at 2 weeks postpartum and keep it till
6 weeks postpartum

For young multiparous women,

- **Fothergill/Manchester procedure** is done that involves
 - i. Amputation of cervix (will lead to incompetent cervix)
 - ii. Plication of Mackenrodt's ligament
 - iii. Preliminary dilatation & curettage
 - Make sure she is not anxious to conceive as the cervix is rendered incompetent by the procedure
 - To prevent the cervical area adhesions, a dilator is placed while healing

For young nulliparous women,

- Various sling surgeries are done
 - i. **Shirodkar's sling** → The isthmus of the cervix is attached to the anterior longitudinal ligament of sacrum
 - ii. **Purandare's sling** → Isthmus of the cervix is attached to rectus sheath
 - iii. **Khanna's sling** → Isthmus of the cervix is attached to ASIS
 - iv. **Virkud's sling** → Purandare's on one side + Shirodkar's on other side

Old women fit for surgery

- **Vaginal hysterectomy + pelvic floor repair**
 - vaginal hysterectomy with pelvic floor repair → Ward Mayo's surgery
 - if cystocele is present → anterior colporrhaphy/kelly's stitch
 - if rectocele is present → Posterior colpoperineorrhaphy

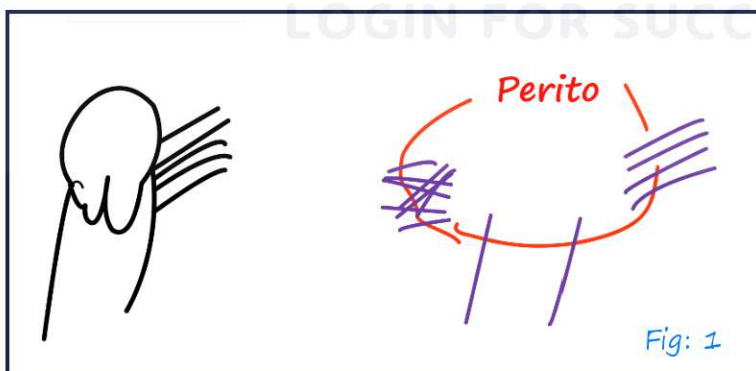
Old women unfit for surgery

- For old woman, who is unfit for surgery, prolapse can be managed by
 - i. pessary
 - ii. **lefort surgery** → Approximate the anterior/posterior walls of vagina

Vault Prolapse

00:14:28

i. MC. Call's Culdoplasty



- Suturing the mckenrodt's ligaments on both sides together with the peritoneum
- Done to prevent vault prolapse post hysterectomy

ii. Sacrospinous Colpopexy

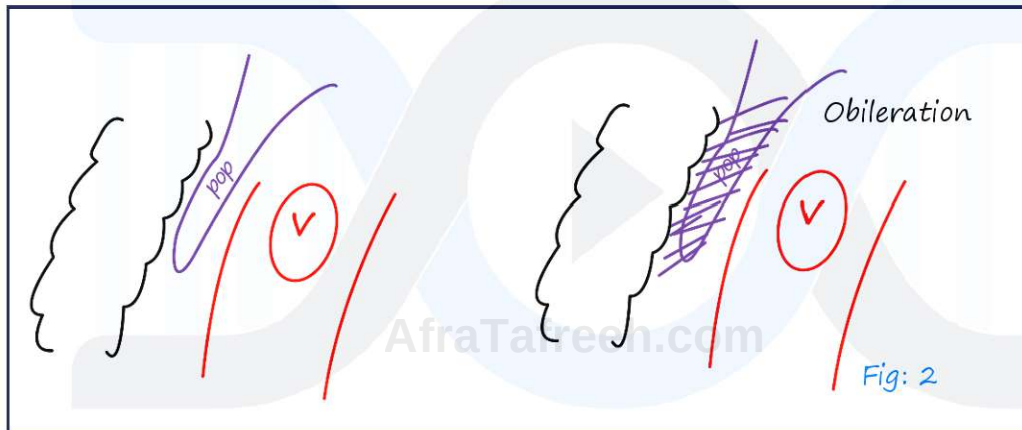
- Attaching the vaginal vault to the sacrospinous ligament
- Done to prevent and treat vault prolapse

iii. Ileococcygeous Colpopexy

- Attaching the vaginal vault to the ileococcygeous muscle

iv. Enterocoele

- Can be Prevented by moscowitz & Halbanz procedures (obliteration of pouch of Douglas)



TUTORIALS

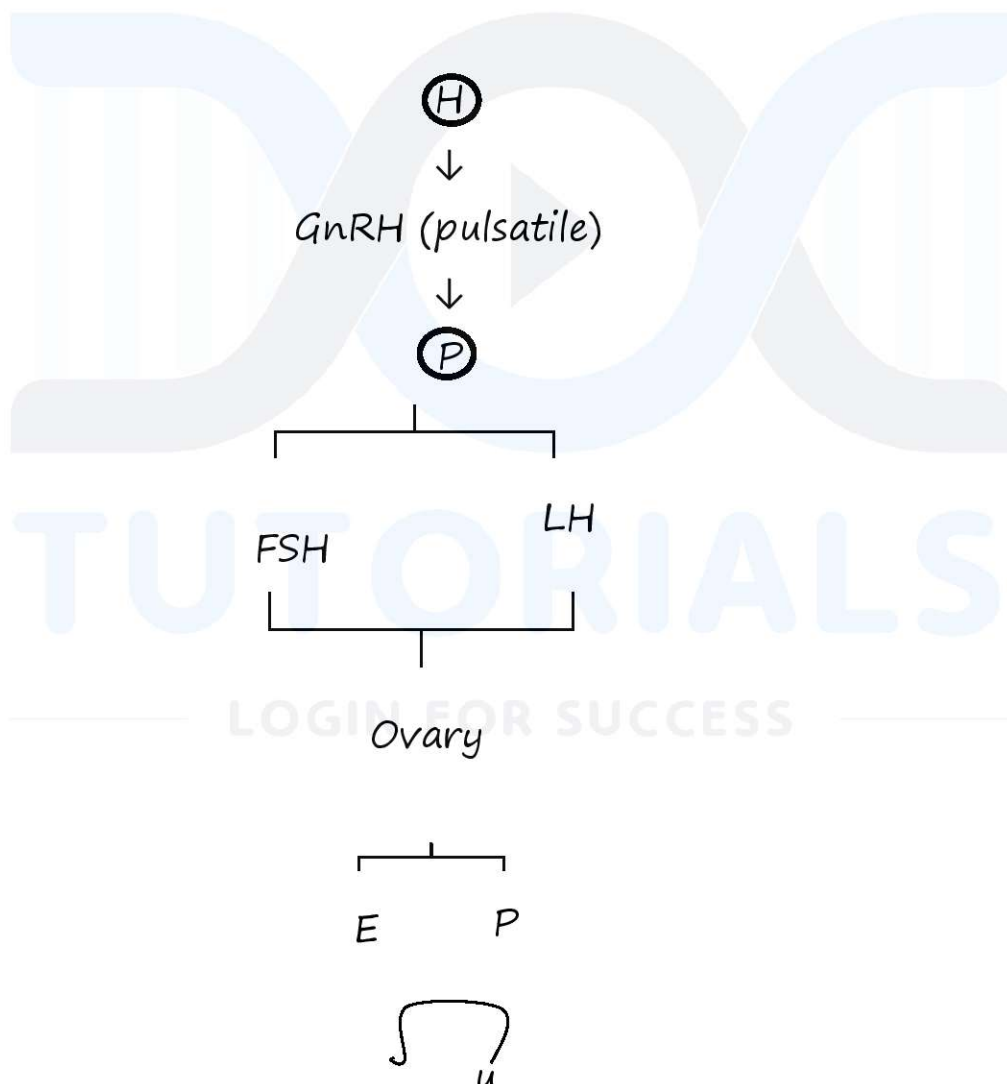
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DRUGS IN GYNECOLOGY

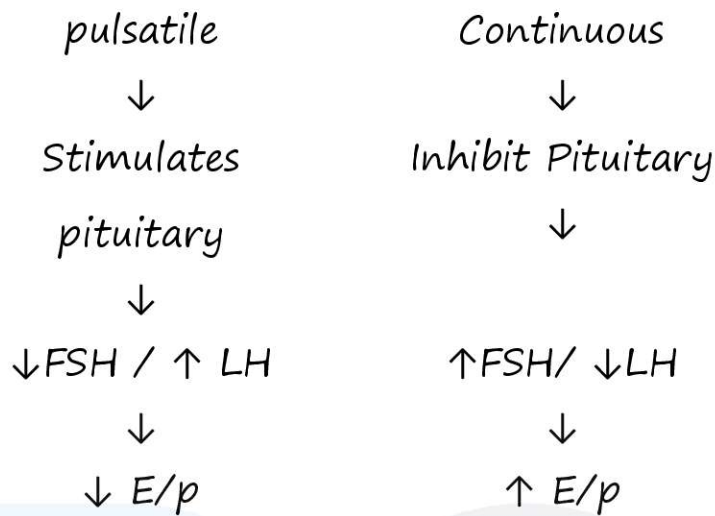
GnRH

00:00:05

- Decapeptide
- Release in a pulsatile fashion from hypothalamus
- $T_{1/2}$ is 2-4 minutes
- Cannot be administered orally (inactivated in gut)
- Recombinant GnRH is not available for therapeutic use



- Note: If GnRH releases in continuous fashion it will down regulate the receptors on pituitary



GnRH Against

00:05:25

- Drugs having GnRH action

Preparation	Dose	Route of Admission
Buserelin	300 mic im/sc daily	im/sc
Naferelin	400 mic/d (200 mic BD)	im
Goserelin	3.6 mg monthly/ 10.8 mg 3 monthly	sc implant im depot
leuprolide	3.75mg monthly	sc im depot

	11.25mg 3 monthly	
Histerelin	50mg yearly	sc implant
triptorelin	3.75 mg monthly 11.25mg 3 monthly	im depot

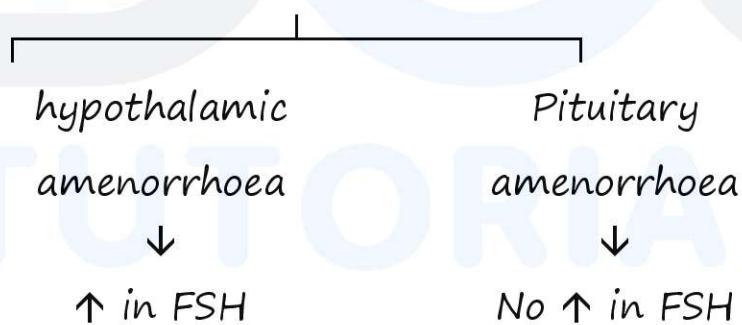
Uses

00:10:50

I. Diagnostic Uses

- i. To differentiate hypothalamic/pituitary amenorrhoea

GnRH stimulation test (pulsatic)



- ii. To differentiate central & peripheral precocious puberty

GnRH stimulation test	
↑ in FSH/LH ↓ Central precocious puberty	No ↑ in FSH/LH ↓ Peripheral precocious puberty

II. Therapeutic uses:

a. Pulsative therapy

i. Hypothalamic amenorrhoea

- IV in low dose
- 2.5 – 5 mg/pulse every 60–90min

ii. Infertility

- Down regulation prior to OI
- OI in hypothalamic dysfunction
- CC resistant PCOS
- Hyperprolactinemia
- A/E multiple pregnancy – 5–8%
- OHSS 1–8%

b. Continuous therapy

- Central precocious puberty
- Endometriosis
- Fibroids
- Abnormal uterine bleeding
- Endometrial ablation (preoperative thinning)
- Hormone responsive breast cancer
- Cryptorchidism & positive cancer in males
- A/E
- Menopausal symptoms (in 95%)
- (can be used only for 3–6 months)
- Hot flushes
- Vaginal dryness

- Insomnia
 - Dyslipidemia
 - Decrease in BMD (osteoporosis)
-
- Initial flare effects lasts for 1 week
 - E/P suppression begins after 1-2 weeks
 - Shrinkage of fibroid starts at 4th week
 - Uterine volume decreases by 40-50% & most of this occurs in 1st 3 months
 - Decrease in fibroid size, pain & bleeding
 - Women usually become amenorrheic

Disadvantages:

- After discontinuation, myoma resumes in 4-10 weeks
- Myoma regrows & attains pretreatment size in 3-4 months

Add Back Therapy

00:32:15

- I. E+P – prevents bone loss & vasomotor symptoms
 equine estrogen – 0.3 to 0.62mg D1-25
 medroxy progesterone acetate 10mg D16-25

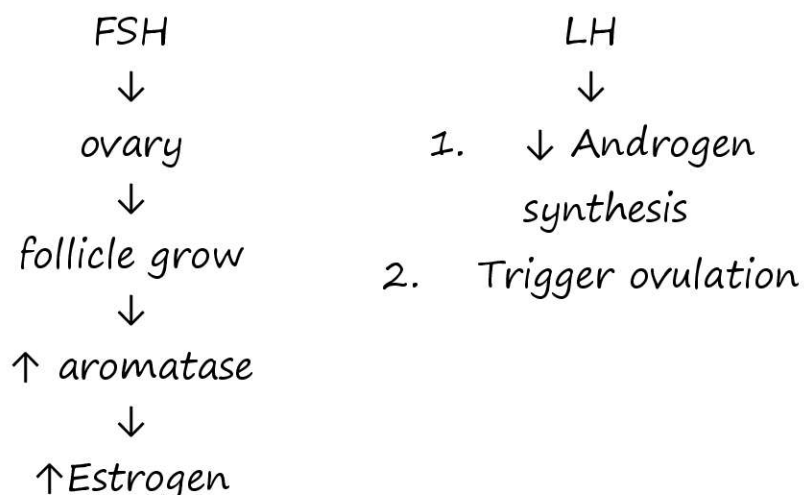
- II. Tibolone } Prevents only bone loss
- Raloxifene }

- Act on GnRh receptors & inhibit them
- They act faster than agonists
- Do not have the initial flare effect (like agonists)
- Used for rapid down regulation (prior to OI)
- Associated with hypersensitivity & not routinely used

Preparations (Daily injectables):

- Cetrorelix – 0.25mg sc (FDA approved for OHSS)
- Ganirelix } SC
- Abarelix }
- Degarelix
- Elagolix – non peptide oral GnRH antagonist
evaluation for endometriosis & fibroids

- FSH – stimulates follicular growth & estrogen production
- LH – Triggers ovulation



Preparation	Dose	Route
Menotropin (HMG) (Human Menopausal gonadotropin)	75U FSH + 75U LH	im
Urofollitropin (U FSH)	75U	sc
Follitropin (r FSH)	50U	sc

- Corifollotropin FSH β sub unit & C terminal peptide of HCG
- Others
- Recombinant LH
- Urinary HCG
- Recombinant HCG
- Uses
 - i. Induction of ovulation & hyperstimulation in ART
 - ii. Hypogonadotropic hypogonadism
 - iii. Clomiphene resistance
 - iv. Unexplained infertility
 - v. Cryptorchidism in males

Uses of HCG:

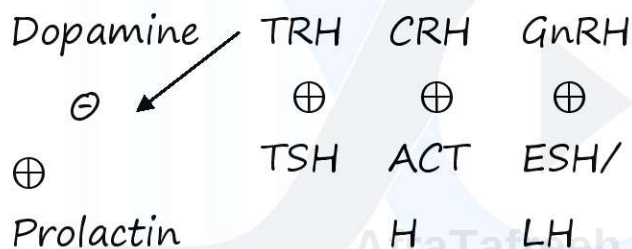
- To trigger ovulation

- Recurrent pregnancy loss
- Lateal support in IVF
- A/E – Multiple preganancy
- OHSS

Prolactin

00:51:23

- TRH stimulates prolactin release
- Dopamine
- Inhibits prolactin release



- Prolactin inhibitors are dopamine receptor aganists
- Ergot alkaloids
- Bromocriptine
- Cabergoline

Bromocriptine	Cabergoline
• Stimulates both D1 & D2 receptors short acting • less effective	• only on D2 receptors long acting • more effective • less side effects

• more side effects (nausea, vomiting, dizziness, nasal stuffiness, hypotension) • No 5HT_{2B} agonistic activity	• 5HT_{2B} agonistic activity • □ risk of valvular heart diseases
--	--

- Can be used in hyperprolactinemia with or without tumor
- For OI in infertility
- To treat galactorrhoea with or without hyperprolactinemia
- In PCOS

Dose	Starting	Maximum
Bromocriptine	1.25mg bed time	5mg twice daily
cabergoline	0.25mg twice weekly	1.5mg twice weekly

- Vaginal administration □ S/E & □ compliance no □ in anomalies/ abortion
- Cabergoline is doc for hyperprolactinemia/prolactinoma even during pregnancy

INSTRUMENTS - I

Sim's Double Bladed Posterior Vaginal Speculum

00:03:11



- Also called duck – bill speculum
- Used to retract the vaginal wall, usually posterior wall
- Used double ended (or) single ended
- If double ended, each end is of different sizes – 26 & 31mm, 31 & 36mm, 36&41mm
- Blades are rounded & atraumatic
- The handle is slightly concave to collect drained blood and secretions
- Advantages
 - It is used to inspect vagina & cervix at a time inside
- Disadvantages
 - Not self retaining
 - Moves with the hand so not suitable for colposcopy
 - Patient needs to be at the edge of table
- Indications

- i. To examine Cx and vagina for discharge, cervicitis, polyps, prolapse, carcinoma, malformations
- ii. To carry out biopsy, D & C, HSG, hysteroscopy, vaginal hysterectomies, colpotomy/culdocentesis
- iii. To examine tears
- iv. To pack uterine cavity.

Sim's Position

00:10:00

- Left lateral position with right knee and thigh drawn up towards the chest, the left arm along the back

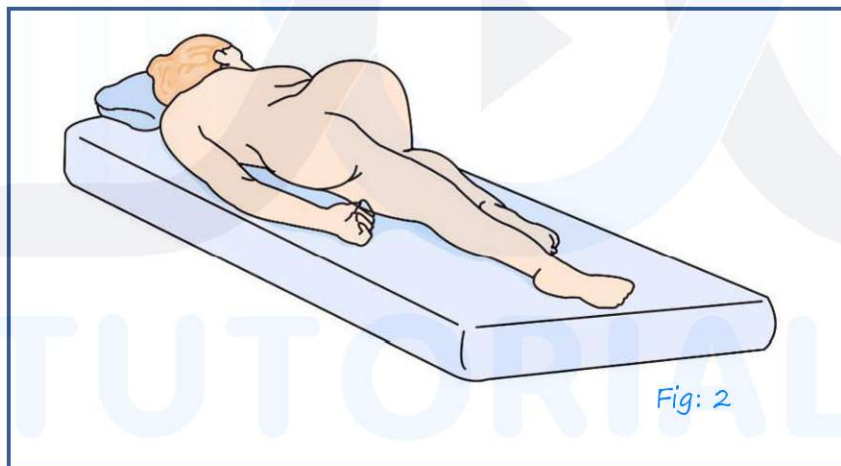


Fig: 2

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Fig: 3

- Indications
 - Cervical and vaginal inspection
 - Perform pap smear, cervical biopsy, colposcopy colpomicroscopy
 - To insert IUCD
- Disadvantages
 - Cover anterior & posterior vaginal wall
 - Decreased maneuverability
 - Less space to perform D & C

What is Transformation Zone:

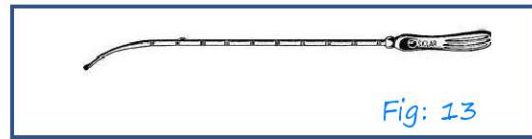
- The place/area between the old and new squamo-columnar junction. It is filled with newly formed squamous cells

Cervical Ectopy:

- The area where squamous cells becomes columnar

Uterine Sound

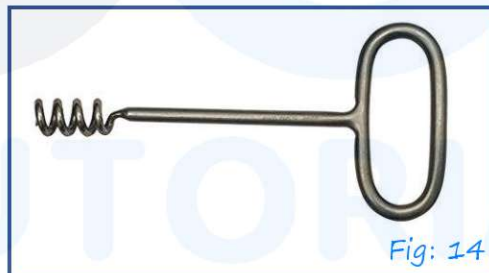
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- Used for
 1. To measure uterocervical length
 2. To measure version of the uterus
 3. To differentiate inversion from prolapse
- It is curved and 45° angulation
- It has graduations

Myoma Screw

00:34:23



- Spiral curved tip with sharp edge

Indications for Hysterectomy:

- Prolapse uterus
- Fibroid uterus
- Adenomyosis
- Genital malignancies
- Dysfunctional uterine bleeding
- Obstetric causes

- Severe PPH
- Rupture uterus

Indications for Myomectomy:

- *Fibroids < 40%*
- *Associated C infertility*
- *Submucous fibroids*
- *Symptomate fibroids*

Kochers Forceps

00:36:44



Fig: 15

- *Used in hysterectomy*

LOGIN FOR SUCCESS